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THE RICE DIET IN AMBULATORY PATIENTS WITH ESSENTIAL HYPERTENSION

A Two-Year Study of 105 Patients

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A YEAR and a half ago an evaluation of the first year's use of the rice diet in hypertension was reported by the Hypertension Clinic of the Massachusetts General Hospital.[1] Our study included only clinic patients, that is, patients living at home, eating with the family group and subject to the usual influences of everyday living. The conditions of this study were different from those reported by Kempner[2-5] and, we submit, in some respects more suitable than his in that most patients must be treated in their homes and at their work with medical supervision through calls at the physician's office or clinic. The present paper reports a second year's study of the rice diet and brings up to date the status of those patients treated in the previous study.

The general conduct of our clinic is as follows: The secretaries of the clinic use an appointment system, which avoids long waits and prevents tension and loss of time and money for the working patient. The patients are seen at fairly frequent intervals (in cases of very restrictive diets, every week), but it is explained carefully that we are personally interested in studying the diet and how it is tolerated, and that we are not alarmed about the patient's hypertensive state. When a patient is first seen in the clinic, a complete history and physical examination are done, and routine blood and urine studies are obtained. In taking the history we are particularly interested in the known duration of blood pressure, duration and description of symptoms, family history (indicating a positive or negative familial tendency to hypertension), age, sex, race, weight and height of the patient. The following studies are obtained:

Examination of the fundi by an ophthalmologist experienced in grading retinal vessels.
Seven-foot heart plate — for size and shape of heart.
Electrocardiogram for hypertensive changes or other abnormalities.
Renal-function tests (Phenolsulfonphthalein test and urine-concentration test).
Intravenous pyelogram, if warranted.
Posture and cold test.
Benzodioxane test.
Chemical studies of serum, including nonprotein nitrogen, cholesterol, sodium and chloride.
Urinary-chloride excretion on normal diet.

During the interval necessary for obtaining these studies the patient is seen several times and evaluated clinically. Control blood pressures are obtained; at each visit the blood pressure is taken three times — on admission, during the interview and just before departure. Often the patient has been seen a number of times in the general Medical Clinic before being referred to the Hypertension Clinic. The control period, therefore, is at least three months and more often several years. After clinical judgment of the patient's status has been made and laboratory data and other studies put in proper perspective, a decision is made concerning the program to be followed — choosing the therapy that seems to offer the most benefit for the individual patient. This may be one of the following:

Conservative medical therapy (weight loss, reassurance, mild sedation and ample psychotherapy).
Drug therapy (at present we are interested in evaluating *Veratrum viride*).
Surgery — either dorsolumbar or dorsal sympathectomy, depending on the age and condition of the patient. We advise surgery frequently in early Grade IV (malignant) cases of hypertension and sometimes in Grade II cases, under age 45.
Diet — We are currently using several types of diet, depending on the needs of the patient; that is, simple reducing, low-sodium and rice diets.

Certain limitations have been placed on our choice of patients for the rice diet, for we find that unless a motivation already exists — either severe symptoms or anxiety concerning blood pressure — the patients do not adhere to this monotonous diet and it is useless to put them on it. We are convinced that the motivation of fear has disastrous results in those patients who adhere strictly to this regimen and even worse effects in those who do not. The patients we have studied on the rice diet are limited to ambulatory patients who are able to

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come to the clinic and who fall into one of the following categories: patients who have developed an intense anxiety concerning their "blood pressure" before we have seen them; patients who are seriously but not acutely ill, showing definite damage to one or more organs; and patients with malignant hypertension who may have progressed too far to be benefited by surgery.

Thus the number of patients is small and represents only 20 per cent of the patients with hypertension treated by us. A relatively large proportion of these patients have severe hypertension.

The procedure followed has been given in the previous report and is summarized briefly as follows: Weekly visits are required, so that we may observe the patients for objective and subjective changes, detect any evidence of sodium depletion, check the weight (if weight loss is too rapid, the caloric intake is increased), check urinary chloride excretion to determine adherence to the diet, obtain blood-pressure readings and encourage the patients when their spirits droop. The length of time that a patient is maintained on the rice diet varies considerably, depending on objective changes such as blood pressure and subjective changes such as improvement in symptoms and toleration of the diet. In general, the time varies from four weeks to four months.

To determine whether the patients are actually adhering to the diet, we have continued the checking of urinary chloride excretion. A change in our program during the second year consisted of the determination of the urinary chloride excretion in the clinic in the presence of the patient, the purpose of the test having been carefully explained. The facial-color changes are quite dramatic. The patients are interested, confess their lapses, are encouraged to adhere strictly and are motivated in their effort to show good results in the next test. When a patient seems to have been strictly on the diet but his urinary chloride excretion is higher than expected, the salt-losing syndrome is suspected and serum-sodium and nonprotein-nitrogen levels are determined. In this way we have recognized poor kidney function that was not apparent at the original renal-function tests. As before, those patients who have urinary chloride excretions consistently below 10 milliequiv. per liter have been included in the study as having been strictly on the rice diet. Those patients with urinary chloride excretions between 10 and 50 milliequiv. per liter are considered to be on a moderately restrictive diet and those with excretions above 50 milliequiv. per liter, for the purpose of this study, are not regarded as representing dietary sodium restriction. Properly, one should compute the chloride excretion for twenty-four hours, but the inaccuracies that arise because the patients fail to measure correctly the total output of urine have led us to use the determination of the chloride on one specimen. Furthermore, it should be noted that if a patient is strictly on the rice diet and his kidney function is good the excretion of chloride in one specimen should be as low as 1 or 2 milliequiv. per liter. Therefore, when we include all those with excretions of 10 milliequiv. or below as adhering strictly to the diet we are probably including some patients with very slight or minor lapses. However, most of the patients considered "strict" have excretions of less than 2 milliequiv. of chloride.

In evaluating the results of our study we have employed the following categories: change in blood pressure, clinical evaluation of change in physical state, subjective change, that is, improvement in symptoms, and change in the various laboratory data.

As in our previous paper, to determine an "improvement" in blood pressure we have placed the blood-pressure recordings in numerical order, choosing the median. The criterion chosen as showing an improvement in blood pressure is a reduction in the diastolic pressure of 20 mm. of mercury. Also as before, we have compared these results

TABLE 1. *Strict Adherers — 1948–1949 Group.*

PATIENT	CONTROL PERIOD	NO. OF WEEKS ON DIET	MEDIAN BLOOD PRESSURES				MEAN ARTERIAL PRESSURE (KEMPNER)*			CHANGE IN WEIGHT
			PREDIET	POSTDIET	CHANGE		PREDIET	POSTDIET	CHANGE	
					systolic	*diastolic*				
										kg.
D. B.	4 mo.	4	188/128	142/110	46	18	158	128	30	−2.8
N. C.	8 yr.	5	200/120	120/86	80	34	156	100	56	−2.1
G. D.	18 mo.	4	186/90	190/100	++	+10	140	141	+1	−3.5
W. F.	1 yr.	4	200/120	201/101	19	19	169	151	18	−0.9
M. G.	10 yr.	8	195/108	162/95	33	12	152	131	21	−2.2
J. G.	14 mo.	6	220/130	190/130	30	0	176	162	14	−2.3
J. K.	2 mo.	9	280/148	260/146	20	2	210	201	9	−3.0
E. M.	13 yr.	8	198/102	160/100	38	2	150	130	20	−7.0
L. P.	56 mo.	7	230/140	198/129	32	11	191	160	31	−5.4
A. T.	3 wk.	18	220/120	200/120	20	0	173	161	12	−6.0
J. B.	3 yr.	2	188/110	150/94	38	16	149	124	25	−2.2
R. T.	6 wk.	4	180/101	140/98	40	3	140	119	21	−6.8
Total improved					1				7	

*Mean arterial pressure = average of all systolic plus average of all diastolic pressures divided by 2.

with those obtained by Kempner's method of determining improvement, that is, using the mean arterial pressure with a reduction of 20 mm. of mercury.

The total number of patients instructed in the use of the rice diet over a two-year period was 105. The number for the first year (1947–1948) was 56, and that for the second year (1948–1949) 49, 8 of whom were on a second rice trial and will not be included in the following summary. Of the total group (two-year period), 28, or 29 per cent, were strict adherents, 35, or 36 per cent, were moderate adherents and 34, or 35 per cent, were delinquents. Seven of the 28 patients (25 per cent) strictly on the rice diet showed improvement in respect to blood pressure (or other categories or both) when evaluated by our method, whereas 17, or 61 per cent, showed improvement when evaluated by Kempner's method. Eight of the 35 patients (23 per cent) considered moderate adherents showed improvement by our method, whereas 18, or 51 per cent, showed improvement when judged by the Kempner criterion. Of the 41 patients treated for the first time during the second year, 12, or 29 per cent, were considered strict adherents, 15, or 37 per cent, moderate and 14, or 34 per cent, delinquents. On the basis of blood pressure in the strict group 1, or 8 per cent, improved by our method and 7, or 58 per cent, improved by Kempner's, whereas in the moderate group 1, or 7 per cent, was improved by our method and 7, or 47 per cent by Kempner's. A detailed description of the blood-pressure changes in the second group will be found in Tables 1 and 2.

A patient may have a marked change in systolic pressure with little or no change in diastolic pressure; this will result in a definite improvement in blood pressure when the criterion of the mean arterial pressure is used. This same patient would not show an improvement in blood pressure if the median diastolic pressure were used. A more graphic description of the difference between the two criteria is shown in Figure 1.

The validity of the criteria that we have used is borne out by the follow-up study of the 1947–1948 group, which shows a much larger percentage of patients who maintained their improvement over a period of two years. We have made definite efforts to obtain a complete follow-up study on the 1947–1948 group, for it is our conviction that the evaluation of any therapy for hypertension is significant only when followed for a sufficiently long period of time to be compared to the material used in the studies of the natural life history and survival of hypertensives.[6-9] Tables 3 and 4 show the data on the two-year follow-up study of the 16 strict and 20 moderate adherents in the 1947–1948 group.

The figures at the foot of Tables 3 and 4 show that by Kempner's method 10 were originally considered improved, but only 3 showed continued improvement after another year of follow-up study. By our method 6 were originally considered improved,

TABLE 2. *Moderate Adherers — 1948–1949 Group.*

PATIENT	CONTROL PERIOD	NO. OF WEEKS ON DIET	MEDIAN BLOOD PRESSURES				MEAN ARTERIAL PRESSURE (KEMPNER)*			CHANGE IN WEIGHT
			PREDIET	POSTDIET	CHANGE		PREDIET	POSTDIET	CHANGE	
					systolic	*diastolic*			*mm. of mercury*	*kg.*
M. A.	30 mo.	5	180/100	160/82	20	18	140	121	19	−3.0
R. C.	2 wk.	9	210/150	200/148	10	2	192	172	20	−8.1
A. C.	3 yr.	11	210/132	190/120	20	12	171	153	18	−3.7
A. D.	17 mo.	14	222/132	207/129	15	2	179	169	10	−0.9
C. G.	18 mo.	23	170/108	160/104	10	4	142	132	10	−4.8
J. H.	17 mo.	10	190/120	176/126	−14	+6	166	148	18	−1.8
F. J.	4 wk.	6	190/120	146/100	54	20	160	125	35	−7.7
L. M.	12 yr.	9	185/135	180/130	5	5	161	152	9	−6.6
R. M.	3 wk.	21	169/118	137/100	32	18	143	120	23	−5.2
S. N.	3 wk.	12	259/140	243/130	16	10	198	187	11	−1.0
J. P.	8 yr.	9	180/130	185/130	5	0	151	159	+8	−6.1
M. Q.	18 mo.	13	218/126	184/118	34	8	172	150	22	−4.0
A. R.	5 yr.	5	210/120	169/120	41	0	166	144	22	−6.2
C. R.	16 wk.	16	192/100	150/100	42	0	150	130	20	−6.8
E. R.	13 yr.	9	210/120	169/109	41	11	168	143	25	−4.5
Total improved					1				7	

*Mean arterial pressure = average of all systolic plus average of all diastolic divided by 2.

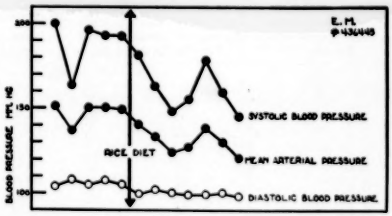

FIGURE 1. *Systolic and Diastolic Pressures Recorded on One Patient.*

The mean arterial tension, which indicates improvement, is also plotted. There is no variation in the diastolic pressure.

and after an additional year of follow-up study 4 had maintained the improvement.

Evaluation of objective changes, other than blood pressure has been difficult, as in most cases the changes were so slight that one could not attribute them to the particular treatment. The definite changes that occurred in the 28 patients who adhered strictly to the rice diet are commented on below. In the examination of the fundi there was regression of hemorrhage, exudate and papilledema in 2 cases out of 28. In 4 cases there was a definite progression in disease. In one case there was tern occurred in 3 of 28. Enlargement of the heart as shown by x-ray examination was present in 25 of 28 patients. In 4 there was a decrease in heart size. However, in 1 of the 4 cases the heart had again become enlarged after two years. On the basis of the urine concentration and phenolsulfonphthalein tests, prior to dieting 15 were considered normal and 13 abnormal; after dieting 3 of the normal patients showed reduced function and 2 of 13 abnormal showed slight improvement.

Subjective improvement is notably difficult to evaluate, as Ayman[10] has so well shown. Most patients feel subjectively improved on any treatment enthusiastically given. It is not surprising, therefore, that most of the patients, whether strict or moderate adherents, reported improvement in their symptoms.

TABLE 3. *Strict Adherers — 1947–1948 Group.*

PATIENT	BLOOD PRESSURES		MEDIAN POSTDIET CHANGES				MEAN POSTDIET CHANGES IN BLOOD PRESSURE (KEMPNER)		
	1948		1949		1950		1948	1949	1950
	FIRST REPORT						FIRST REPORT		
	systolic	*diastolic*	*systolic*	*diastolic*	*systolic*	*diastolic*			
G. A.	42	17	36	20	24	16	27	25	16
F. B.	25	5	25	5	Deceased	Deceased	16	16	Deceased
S. B.	10	2	10	4	10	0	8	14	8
W. D.	30	10	0	0	Deceased	Deceased	17	+1	Deceased
F. J.	22	13	22	15	12	13	23	25	19
I. K.	30	0	30	0	20	0	20	17	13
W. K.	24	0	24	0	14	+2	12	11	4
M. Mc.	35	22	35	22	+35	+10	30	30	15
C. N.	34	30	30	27	20	20	36	32	25
D. P.	25	10	15	10	+7	4	18	16	0
A. S.	25	4	25	0	−33	+9	15	14	11
T. S.	60	26	60	22	50*	20	28	26	23*
D. S.	30	18	30	18	29	23	27	27	26
V. V.	60	36	53	35	30	22	39	37	18
J. W.	60	30	Deceased	Deceased	Deceased	Deceased	45	Deceased	Deceased
P. Z.	30	20	10	6	20	10	23	8	14
Total improved	6		5		4		10	7	3

*Two trials.

TABLE 4. *Moderate Adherers — 1947–1948 Group.*

PATIENT	BLOOD PRESSURES		MEDIAN POSTDIET CHANGES				MEAN POSTDIET CHANGES IN BLOOD PRESSURE (KEMPNER)		
	1948		1949		1950		1948	1949	1950
	FIRST REPORT						FIRST REPORT		
	systolic	*diastolic*	*systolic*	*diastolic*	*systolic*	*diastolic*			
A. A.	20	10	20	0	Deceased	Deceased	16	13	Deceased
C. B.	20	12	10	5	+40	+22	13	8	+30
P. B.	43	14	40	10	Deceased	Deceased	25	24	Deceased
O. C.	2	8	2	8	+38	+10	8	8	+21
N. C.	20	12	20	12	−10	+2	16	16	1
H. E.	50	22	50	20	40	10	34	32	23
J. F.	−1	+1	Deceased	Deceased	Deceased	Deceased	4	Deceased	Deceased
G. F.	+1	−10	+9	−10	+20	−4	10	6	+8
M. H.	20	10	20	10	+37	−12	28	28	+2
O. J.	34	20	26	20	43	30	30	27	38
K. Mc.	12	7	12	7	−	+3	18	18	9
M. Mc.	54	6	30	2	4	1	21	9	0
T. Mc.	36	2	20*	0	36*	19	19	22*	27*
M. M.	10	+10	Deceased	Deceased	Deceased	Deceased	2	Deceased	Deceased
L. M.	50	20	50	12	30	0	23	20	11
M. R.	72	16	52	10	+26	0	37	33	+6
T. S.	19	17	19	17	+18	−11	10	10	+8
P. S.	51	18	48	20	14	4	36	25	13
D. W.	+8	10	Deceased	Deceased	Deceased	Deceased	+2	Deceased	Deceased
C. W.	25	10	25	8	−5	+4	24	25	5
Total improved	2		3		1		9	8	4

*Patient had had two cerebral vascular accidents.

a change from an arrhythmia to a normal sinus rhythm. In the majority of cases, however, the abnormal electrocardiographic evidence consisted of the so-called hypertensive pattern; in this there was no change in 4. A reversion to a normal pat-

Discussion

An evaluation of any therapy for hypertension must make use of a well supervised control group on no therapy or on conservative medical therapy for comparison. In two years' time (which must be taken as a brief and preliminary follow-up in this disease) 2, or 12.5 per cent, of the strict group had maintained improvement in blood pressure by Kempner's criterion; 3, or 19 per cent, of the same group had died; in 9, or 56 per cent, the disease had progressed; and in 4, or 25 per cent, the disease had seemingly remained stationary. In a random group of patients treated on a conservative medical program, we could easily match these results, unless the group were weighted with cases of malignant hypertension.

The factors in favor of the use of the rice diet are that it offers a very definite form of therapy for those patients who have extreme blood-pressure anxiety, that it teaches self-discipline to patients who can accept it and that it is easy to teach and simple to follow.

The factors against its use are several. The diet is by no means innocuous; it is lethal if the patient is not carefully watched. The increased time needed for observation puts a heavy load on the doctor and increases the cost to the patient, because of weekly visits. The diet is so monotonous that in some patients it has seemed responsible for recurrent depressions and in others for overt hostility such that we have been unable to persuade patients to return to the clinic.

We are at present planning a dietary program for the next several years, using a diet that has a normal protein content, a very low fat content, a low salt content and carbohydrates higher than normal unless it is desirable for the patient to lose weight. We expect this diet to be acceptable to the patient; it has definite advantages, moreover, from a nutritional point of view. We hope to follow patients on this diet for an equivalent time, and compare them with our results on the rice diet.

Summary

An evaluation is presented of two groups of outpatients on the rice diet. The study of the first group was started in 1947; a further follow-up study of this group is reported. The study of the second group was started in 1948; the results obtained in this group are shown here for the first time. It is our opinion at this point that the rice diet is unpleasant and expensive for the patient, very demanding on the doctor and potentially dangerous. In ambulatory patients it produces no better results than more conservative medical therapy. Nevertheless, the rice diet in certain patients severely ill with hypertension apparently causes dramatic reversal of the disease process. It seems to us that to justify so rigorous a treatment it must be matched by the actual or impending rigors of the disease.

References

1. Loofbourow, D. G., Galbraith, A. L., and Palmer, R. S. Effect of rice diet on level of blood pressure in essential hypertension. *New Eng. J. Med.* 240:910-914, 1949.
2. Kempner, W. Treatment of kidney disease and hypertensive vascular disease with rice diet. *North Carolina M. J.* 5:125-133 and 273, 1944; 6: 61 and 117, 1945.
3. *Idem.* Some effects of rice diet treatment of kidney disease and hypertension. *Bull. New York Acad. Med.* 22:358-370, 1946.
4. *Idem.* Treatment of hypertensive vascular disease with rice diet. *Am. J. Med.* 4:545-577, 1948.
5. *Idem.* Treatment of heart and kidney disease and of hypertensive and arteriosclerotic vascular disease with rice diet. *Ann. Int. Med.* 31:821-856, 1949.
6. Bechgaard, P. Arterial hypertension: follow-up study of one thousand hypertonics. *Acta med. Scandinav.* Supp. 172:3-358, 1946.
7. Palmer, R. S., Loofbourow, D. G., and Doering, C. R. Prognosis in essential eight-year follow-up study of 430 patients on conventional medical treatment. *New Eng. J. Med.* 239:990-994, 1948.
8. Blood, D. W., and Perera, G. A. Hypertensive vascular disease: duration of life in selected series. *Am. J. Med.* 4:83-88, 1948.
9. Burgess, A. M. Excessive hypertension of long duration. *New Eng. J. Med.* 239:75-79, 1948.
10. Ayman, D. Evaluation of therapeutic results in essential hypertension: interpretation of symptomatic relief. *J. A. M. A.* 95:246-249, 1930.

UNTOWARD REACTIONS TO TESTS FOR EPINEPHRINE-SECRETING TUMORS (PHEOCHROMOCYTOMA)*

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SAN FRANCISCO

THE recent development of pharmacologic tests for epinephrine-secreting tumors[1-4] has increased the interest in this field so that in many institutions such procedures are done almost routinely on nearly all patients with hypertension.

The severe and uncontrollable, but transient, hypertension that may occur after the intravenous administration of histamine in patients with pheochromocytoma has been well described.[5] Tetraethylammonium bromide has been proposed as a substitute test substance, primarily because the hypertension following its administration in patients with pheochromocytoma may be effectively countered by placing the patient in a sitting position.[3] At least one death due to tetraethylammonium-chloride sensitivity has been recorded,[6] and others probably have occurred.

The Council on Pharmacy and Chemistry of the American Medical Association authorized the publication of a report[7] stating that the use of one of the adrenolytic substances, piperidylmethyl benzodioxane — hereinafter referred to as 933F§ — was "promising and evidently safe." A few instances have been reported of severe toxicity with the use of adrenolytic agents of the benzodioxane series. The hypertensive rise that follows intravenous injection is a well known property of the benzodioxanes.[8, 9] Mild to moderate rises in systolic blood pressure often occur after the administration of 933F to patients with essential hypertension. Drill[10] records excessive hypertensive reactions in two patients tested with 933F in a dose of 10 mg. per square meter of body surface. One other patient had a convulsive seizure after intravenous injection of 933F, in a dose of 10 mg. per square meter of body surface, associated with a rise of blood pressure from 160/92 to 190/110.[11] The reaction was attributed to the sympathomimetic action of 933F in a susceptible patient.

This report deals with 4 separate cases of severe untoward reactions in four patients, 2 following the use of 933F, 1 after tetraethylammonium bromide and the other after Dibenamine (N,N-dibenzyl-beta-chlorethylamine).¶ It is evident that these agents cannot be used for such tests as a routine office procedure without recognizing that an occasional untoward reaction may be encountered.

METHODS

Fifty-six separate tests were done on 18 hypertensive and 3 normotensive patients suspected of having pheochromocytoma, after careful selection from a larger group of patients. The test substances were injected intravenously through a three-way stopcock incorporated into an infusion arrangement. The injection time for 933F and tetraethylammonium bromide was two minutes. The Dibenamine was added directly to 500 cc. of isotonic saline solution and infused over a two-hour period. All subjects were supine under resting but not basal conditions. Frequent blood-pressure measurements by the standard arm-cuff technic were taken from one-half to two hours prior to administration of the test substance to determine the base-line pressures. More frequent pulse and blood-pressure recordings were made at appropriate intervals during and after injection of the test material until both had returned approximately to the base-line levels.

CASE REPORTS

CASE 1. M.B., a 33-year-old housewife, complained of severe occipital headaches of seven years' duration and exertional dyspnea of increasing severity with excessive fatigue for two years. She delivered a full-term normal child at the age of 25, at which time her blood pressure was normal. An insurance examination shortly thereafter revealed hypertension. The patient had been thoroughly studied at the age of 31 and was advised to undergo a sympathectomy, which she refused.

Physical examination revealed a blood pressure of 245/130. There was a right facial palsy of three days' duration. Ophthalmoscopic examination showed marked arterial-venous nicking and "silver wire" arteries (Keith-Wagener-Barker II). Breath sounds were decreased throughout both lung fields. The left border of cardiac dullness was 1 cm. lateral to the left midclavicular line in the fifth intercostal space. The first and second mitral sounds were loud and snapping. The pulmonic second sound was louder than the aortic second sound and there was a Grade II precordial systolic murmur, loudest at the base. The liver was not enlarged; there was no peripheral edema. The blood count was within normal limits. Urinalysis revealed a +++ albumin, a specific gravity of 1.017 and a protein of 1.3 gm. per twenty-four hours. An Addis count showed 300,000 casts, 65 per cent hyaline, 35 per cent granular; 10,500,000 red blood cells and 142,000,000 white blood cells. An electrocardiogram showed an abnormal record characteristic of left-ventricular hypertrophy. The nonprotein nitrogen was 40 mg. per 100 cc. and the phenolsulfonphthalein excretion was 80 per cent in two hours.

In order to rule out the possibility of a pheochromocytoma, a Benzodioxane test was done (Fig. 1). The patient was to have received 10 mg. per square meter of body surface, or a total of 16 mg. Within thirty seconds after the injection of 933F had started, she appeared flushed and complained of marked weakness, so that further administration of the

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§Kindly supplied by Merck and Company, Rahway, New Jersey, as Benodaine.

¶Kindly supplied by Givaudan-Delawanna, Inc., New York.

agent was halted. Only 3 mg. of 933F had been given. Immediately thereafter the patient went into a shock-like state, with cold, clammy extremities, vertigo and an imperceptible pulse and blood pressure (Fig. 1). During the initial period of injection she said that she felt as if she were dying. This was followed within sixty seconds by a sharp rise in blood pressure to 290/130, flushing, nausea and a throbbing headache, which she said "felt as if it were going to blow off the top of my head." The blood pressure and pulse returned to the base-line gradually after thirty minutes. To establish this reaction more firmly as a sensitivity to 933F, 5 mg. of 933F was injected thirty minutes later, with a similar et

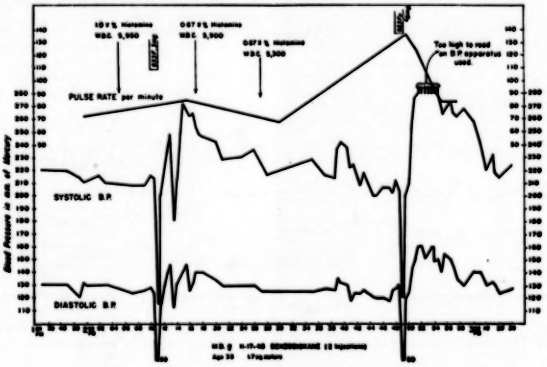

FIGURE 1. *Case 1, M. B.*

Note marked hypotension toward end of administration of 933F on both occasions followed by prompt and excessive rise in blood pressure. The usual dose of 933F would have been 16 mg., but the patient received only 3 and 5 mg., respectively. The blood histamine values indicated by the arrows should be disregarded, as they pertain to another investigation.

more violent reaction (Fig. 1). The pulse rose to 136, and the systolic blood pressure exceeded 300 mm. of mercury. The throbbing headache was almost unbearable; the severe flush-

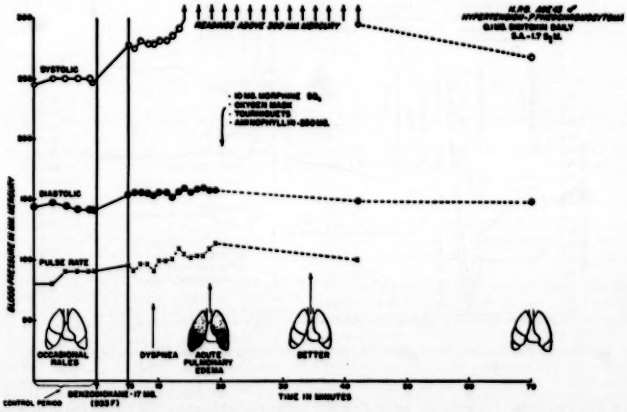

FIGURE 2. *Case 2, H. P. Q.: Development of Acute Pulmonary Edema after Intravenous Administration of 933F (17 mg.).*

ing was followed by profuse diaphoresis. The blood pressure returned to its base-line again in thirty minutes, at which time all the patient's symptoms had disappeared. It was

believed that the patient might have died if the full dose of 16 mg. of 933F had been given.

After the patient's discharge from the hospital, her hypertension became progressively more severe, and she died ten months later.

CASE 2. H. P. Q., a 43-year-old surveyor, was known to have had intermittent hypertension for at least twenty years.

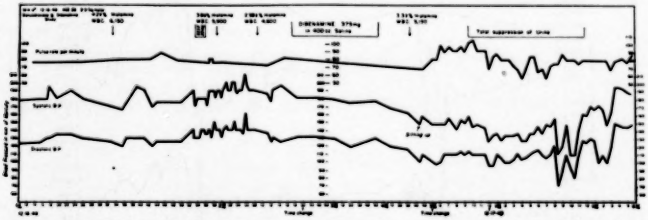

FIGURE 3. *Case 3, M. H. Anuria for Fourteen Hours after Intravenous Infusion of Dibenamine. The period of urinary suppression coincides with the decrease in pulse pressure.*

His recent complaints were of exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea, productive cough with pink frothy sputum, extreme fatigue and anorexia, all of six weeks' duration. He had experienced severe visual impairment, particularly in the left eye, in the past three weeks. He had lost 20 pounds in the past year. Immediately prior to admission he had been digitalized, with considerable improvement in his symptoms.

Physical examination revealed a blood pressure of 238/154 (left arm sitting). Ophthalmoscopic examination revealed bilateral papilledema, generalized and focal arterial narrowing, cotton-wool patches and numerous hemorrhages (K.W.B.IV). There was an exudate in the left fundus, inferior to the disk. Rales were heard posteriorly at both lung bases. The left border of cardiac dullness extended 12 cm. to the left of the midsternal line in the fifth intercostal space. The aortic second sound was louder than the pulmonic second sound, with a Grade II aortic systolic murmur. The liver was not felt, but the spleen was palpable 2 cm. below the left costal margin on deep inspiration. There was no clubbing, cyanosis or edema.

Electrocardiogram showed an abnormal record characteristic of left ventricle hypertrophy; an x-ray film showed cardiac enlargement consistent with left-ventricular hypertrophy. Urinalysis revealed a specific gravity of 1.017, +++ albumin, 6 to 8 red blood cells per high-power field, 2 to 4 white blood cells per high-power field and 4 hyaline and 22 granular casts per low-power field in the sediment. Phenol-

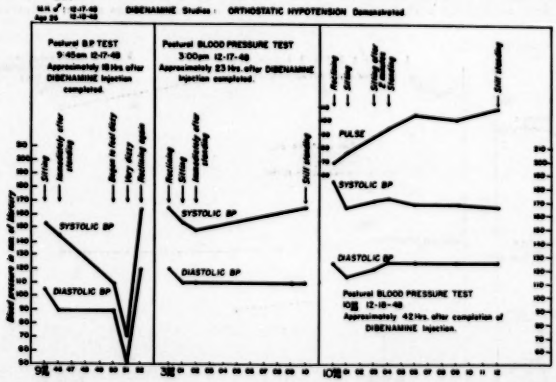

FIGURE 4. *Response to Erect Position Following the Dibenamine Test on Patient M. H.*

sulfonphthalein excretion was 47 per cent in two hours; creatinine clearance was 47 cc. per minute.

The diagnosis was malignant hypertension due to chronic glomerulonephritis with congestive failure, hypertensive encephalopathy and retinopathy.

In order to rule out the possibility of a pheochromocytoma, a Benzodioxane test was performed. After a stable thirty-minute control period, 17 mg. of 933F (10 mg. per square meter of body surface) was administered intravenously.

From a base-line of 250/145, his blood pressure rose above 300/160 within fifteen minutes after the injection (Fig. 2). The pulse rate rose from 85 to 115. The patient became dyspneic, orthopneic and cyanotic. He perspired heavily and complained of sharp precordial pain. Moist rales were heard over both lung fields; an unequivocal diagnosis of acute pulmonary edema was evident. The patient was treated promptly with oxygen, aminophylline, morphine and rotating tourniquets on the extremities. The lungs were clear within forty-five minutes and the patient was completely free of symptoms three and a half hours later.

The patient was subsequently discharged on digitalis and a low-salt diet but soon returned, again in congestive failure.

Case 3. M. H., a 26-year-old student veteran, was admitted to the hospital with a diagnosis of essential hypertension. He was asymptomatic. His hypertension had been discovered two years previously during a routine discharge hours; diuresis corresponded to the time when his blood pressure again began to approach base-line levels (Fig. 3). Postural hypotension was noted eighteen hours after the injection, was less marked at twenty-three hours and had disappeared forty-two hours later (Fig. 4).

Case 4. M. T., a 66-year-old woman, had a history of hypertension for fifteen years. She experienced transient episodes of dizziness of increasing frequency and severity during the previous three years. The recent episodes were so sudden in onset that her family refused to allow her to go out alone. Exertional dyspnea was present during this time, with dependent ankle edema during the past year. She was referred for observation of a possible pheochromocytoma and was studied with histamine, 933F and tetraethylammonium bromide. The reaction occurred with the latter substance.

Physical examination revealed a blood pressure of 270/140. Examination of the fundi showed a K.W.B. II. The heart

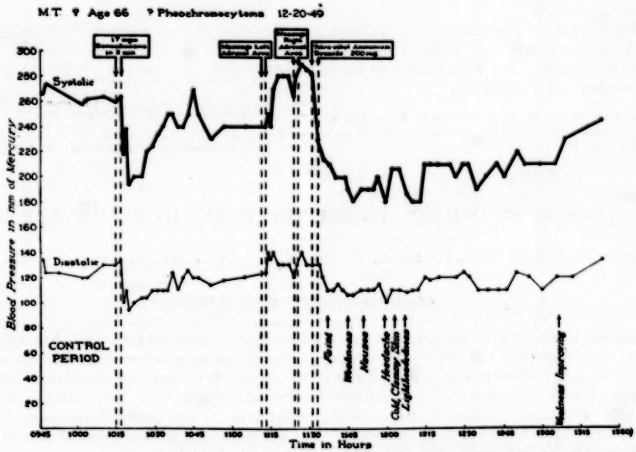

Figure 5. *Marked Hypotension during and following Administration of Tetraethylammonium Bromide (200 mg.).*

Although it is not apparent in the illustration, ten minutes elapsed between the end of massage of the adrenal area and the beginning of the injection.

examination from the Army. He had been studied at the Regional Veterans' Administration Hospital and was admitted for Benzodioxane and Dibenamine studies to rule out a possible pheochromocytoma.

At physical examination, aside from a blood pressure of 180/125 (right arm recumbent) and a bilateral varicocele, the findings were completely normal. Routine urinalysis and blood count were also within normal limits.

On the day after admission 20 mg. of 933F was given intravenously, producing a peak rise of blood pressure from 185/145 to 215/168. A blood pressure of 180/130 was regained within eighteen minutes (Fig. 3). The patient was then given intravenously 375 mg. of Dibenamine (5 mg. per square meter of body surface) in 400 cc. of isotonic saline solution over a period of one hour and forty-five minutes. One hour after completion of the injection the blood pressure began to drop, reaching a low of 110/70 in seventeen hours after completion of injection. It returned to the base-line blood pressure approximately twenty-four hours after injection. During the period of lowest blood pressure there was a total suppression of urine lasting approximately fourteen was enlarged moderately to the left, with a prominent apical impulse. The aortic second sound was louder than the pulmonic second sound, and there was a soft blowing systolic murmur.

After massage of both adrenal areas, 200 mg. of tetraethylammonium bromide was given intravenously over a two-minute period (Fig. 5). Immediately after the injection, the blood pressure began to fall. The patient promptly complained of light-headedness, dizziness and weakness, which became progressively severe as the blood pressure continued to fall, reaching its nadir fifteen minutes after the drug was given. The blood pressure did not begin to rise for an additional ninety minutes; it did not return to the base-line level for three hours. The patient continued to complain of weakness, faintness, nausea and vertigo, which diminished gradually as the blood pressure rose. She was able to leave the ward four hours after the drug was administered, although still weak and upset by the severe reaction; she did not fully recover until thirty-six hours later.

Summary

Untoward severe but nonfatal reactions were encountered in 4 patients during a series of 56 tests on 21 patients suspected of having pheochromocytoma. The first patient developed a rise in blood pressure approaching and exceeding 300 mm. of mercury immediately after the administration of 3 and 5 mg. of 933F. The second patient received 17 mg. of 933F and promptly developed acute pulmonary edema. The third patient developed anuria for fourteen hours following the use of Dibenamine. The fourth patient had a marked hypotensive reaction after a test with tetraethylammonium bromide. The relatively high incidence of toxic reactions in this series is probably due to the selected group of patients studied.

Pharmacologic tests for pheochromocytoma with adrenolytic substances are not without danger; caution must be duly exercised with regard to toxic reactions when they are undertaken.

The authors wish to thank Drs. Lawrence Feigenbaum and Nobuyuki Kawata for their permission to report the data obtained on Case No. 2.

References

1. Roth, G. M., and Kvale, W. F. Tentative test for pheochromocytoma. *Am. J. M. Sc.* **210**:653-660, 1945.
2. LaDue, J. S., Murison, P. J., and Pack, G. T. Use of tetraethylammonium bromide as diagnostic test for pheochromocytoma. *Ann. Int. Med.* **29**:914-921, 1948.
3. Goldenberg, M., Snyder, C. H., and Aranow, H., Jr. New test for hypertension due to circulating epinephrine. *J. A. M. A.* **135**:971-976, 1947.
4. Spear, H. C., and Griswold, D. Use of Dibenamine in pheochromocytoma. *New Eng. J. Med.* **239**:736-739, 1948.
5. Roth, G. M., and Kvale, W. F. Pharmocologic tests as aid in diagnosis of pheochromocytoma. *Mod. Concepts Cardiovas. Dis.* **18**:41, 1949.
6. Lasser, R. P., Rosenthal, N., and Loewe, L. Death following use of tetraethylammonium chloride. *J. A. M. A.* **139**:153, 1949.
7. Grimson, K. S., Hendrix, J. P., and Reardon, M. J. Newer adrenolytic, sympatholytic and ganglionic blocking drugs. *J. A. M. A.* **139**:154, 1949.
8. Handovsky, H. Sur l'effet central du diéthylaminométhyl-3-benzodioxane (F 883). *Compt. Rend. Soc. de biol.* **118**:1245, 1935.
9. Cahill, G. F., and Aranow, H., Jr. Pheochromocytoma: diagnosis and treatment. *Ann. Int. Med.* **31**:389-404, 1949.
10. Drill, V. A. Reactions from use of Benzodioxane (933 F) in diagnosis of pheochromocytoma. *New Eng. J. Med.* **241**:777-779, 1949.
11. Green, D. M., and Peterson, E. M. Hypertensive encephalopathy after administration of Benzodioxan. *J. A. M. A.* **142**:408, 1950.

STELLATE BLOCK AS AN ADJUNCT TO THE TREATMENT OF PULMONARY EMBOLISM*

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FRAMINGHAM, MASSACHUSETTS

MUCH interest has been evinced in recent years in the manifestations of thromboembolic disease. A voluminous literature has appeared concerning the use of anticoagulants and femoral-vein ligation. These measures, however, have been directed primarily toward preventing pulmonary embolism rather than at influencing the immediate effects of the emboli themselves, and little progress has been made in augmenting the meager list of measures that are of use in the treatment of the acute phase of the condition.

The methods of immediate treatment can be divided into operative technics and all others, including the use of drugs, general supportive measures and minor surgical procedures. As regards the former, the Trendelenburg[1] procedure for the prompt removal of a pulmonary embolus is a logical way to combat the lethal mechanisms of the asphyxia, failure of the right heart or reduced effective blood volume that may ensue.[2, 3] This operation, however, is a major undertaking and has seldom proved either practical or successful.[4, 5] Since surgery seems to offer so little hope of success in the early treatment of the condition, if progress is to be made we must turn to the use of drugs such as papaverine or atropine or other measures that will improve the deranged physiology.

It is not the purpose of this paper to review the varied and imperfectly understood pathologic background of thromboembolic disease but rather to discuss and present clinical data concerning the use of stellate block as an adjunct to the immediate treatment of this frequently fatal condition. Leriche,[6] in 1937, was the first to advocate the procedure in the treatment of pulmonary embolism but, largely because of the earlier difficulties in technic, it has been used relatively infrequently, and has not been given an adequate therapeutic trial. The technic of stellate block, using the anterolateral approach, is simple, may be life-saving and is attended in general by no untoward sequelae. We believe that our results, as well as those of others,[7] with injection of procaine into the stellate ganglion warrant its further trial in the early treatment of pulmonary embolism.

The severity of the symptoms of this condition may be out of proportion to the size and number of blood vessels occluded,[8-11] and the outcome does

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not necessarily depend on the size of the obstruction.[8] Neuhof[8] recently reviewed 88 fatal cases and could find "no constant relationship between the degree, location and extent of the pulmonary embolism and the clinical syndrome produced." Leriche and his co-workers[8] found that in 30 per cent of 225 fatal cases only one branch of a lobar artery or one of several lobar arteries was blocked. They believe that death in this group was due to reflex sympathetic pulmonary vasoconstriction and that stellate-ganglion block might have been life-saving. In 35 cases of fatal pulmonary embolism deTakats[9] found only minor pulmonary arterial obstruction in 7. He reasoned that the deleterious reflexes initiated at the embolic site precipitated death in these patients.

Vascular spasm has been demonstrated in peripheral arteries distal to the occlusion of the main arterial pathway.[12] It has also been shown that procaine sympathetic ganglion block or ganglionectomy will prevent vasospasm accompanying arterial occlusion. DeBurgh Daly and his collaborators[10, 11] have pointed out the complexity of the physiology of the interrelated bronchial and lesser pulmonary circulations and have studied the potent sympathetic vasoconstriction that can occur in the pulmonary vascular tree. This spasm of both arteries and bronchi may be extensive and may involve far more than the restricted pulmonary area ordinarily supplied by the occluded vessel. Such spasm can be released by stellate-ganglion block.[6, 9]

Not only are the lungs involved in the reflexes initiated by the embolism but the heart and coronary vessels, because of the intimate association of the innervation of lungs and heart, also share in these changes.[13]

Authors[14, 15] agree that the extrinsic nerve supply to the lungs is derived from the vagus and sympathetic trunks, from which fibers continue on the posterior surface of the hilum to form the posterior pulmonary plexus. The exact distribution of sympathetic and parasympathetic fibers from the posterior pulmonary plexus to the vessels and bronchi of the lung is not known. It seems true, however, from the evidence of several authors, that the vagus and sympathetic trunks contain both afferent and efferent fibers and that normal vascular and bronchial tone depends on a balance between them. The pulmonary embolus initiates reflexes that alter this balance. Probably some of the preganglionic thoracic sympathetic nerves have their synapses at the level of their origin and pass directly to the lung. Procaine infiltration of the stellate ganglion will not affect these fibers but will block the postganglionic sympathetic nerves from the thoracic segments of the cord that have their synapses in the stellate or the middle cervical ganglia. Such a stellate-ganglion block on the apparently affected side alone will interrupt sufficient pathways to alter most of the deleterious reflexes caused by the embolus.

Anatomic dissections[16] have shown that the distance between the middle of the transverse process of the sixth cervical vertebra and the upper pole of the stellate ganglion averages 4 cm. and that the distance from the lateral margin of this process to the sympathetic chain is 0.5 cm. (Fig. 1). The fascial

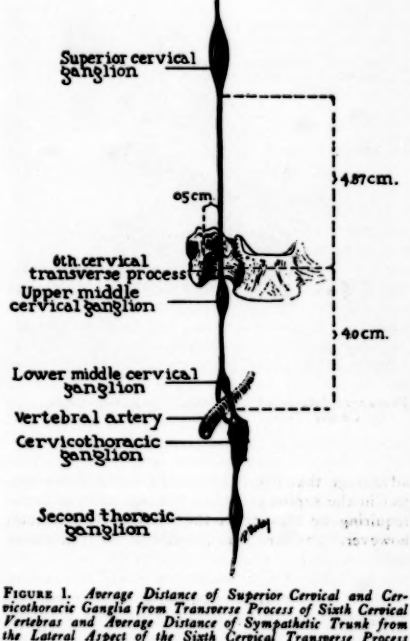

FIGURE 1. *Average Distance of Superior Cervical and Cervicothoracic Ganglia from Transverse Process of Sixth Cervical Vertebras and Average Distance of Sympathetic Trunk from the Lateral Aspect of the Sixth Cervical Transverse Process (Kirgis, H. D., and Reed, A. F., Ann. Surg. 128:101-109, 1948; by permission of the publisher).*

planes of the neck in this area are such that 10 cc. of a 1 per cent solution of procaine placed on or anterior to the anterior prominence of the sixth cervical vertebra will diffuse forward and downward to infiltrate the cervical sympathetic chain and the stellate ganglion (Fig. 2). A Horner syndrome will result almost immediately, signifying the block of the cervical sympathetic fibers. With downward diffusion of the anesthetic agent to involve the stellate ganglion there is warming of the fingers on the blocked side. Our method of performing the block is a modification of the descending-infiltra-

tion technic of de Sousa Pereira[17] and is essentially the same as that described by Caldwell, Broderick and Rose[18] (Fig. 3). This method largely obviates complications such as puncture of the pleura, subarachnoid space and great vessels, which are more likely to occur with other technics. It has the further advantage that it can be carried out with the subject in the supine or sitting position, thus as a rule requiring no moving of the patient. It does not, however, obviate the possibility of temporary paralysis of the recurrent laryngeal or cardiac-accelerator nerves on the same side, and we therefore do not believe that bilateral stellate block should be carried out. We have had the opportunity to perform unilateral stellate-ganglion block on 4 patients with pulmonary embolism. There was dramatic relief of symptoms in 3.

Brief summaries of these cases follow (Table 1).

FIGURE 2. *Relation of the Cervical Sympathetic Chain to the Carotid Tubercle and Surrounding Structures.*

FIGURE 3. *Technic of Stellate Block by the Anterolateral Approach with the Left Index Finger Palpating the Carotid Tubercle of the Sixth Cervical Vertebra and Displacing the Sternocleidomastoid Muscle and Carotid Sheath Anteriorly.*

The needle is introduced anterior to the tubercle in a slightly caudal direction, and 10 cc. of a 1 per cent solution of procaine is injected.

CASE REPORTS

CASE 1. (CVAH, 13605). N. M., a 32-year-old man, was admitted to Cushing Veterans Administration Hospital for the first time on December 24, 1948, with a chief complaint of bloody diarrhea of three months' duration. Physical examination was negative aside from inflamed granular mucosa of the rectum, seen on proctoscopy. A diagnosis of ulcerative colitis was made. On a medical regimen the patient failed rapidly; an ileostomy was therefore performed on January 19, 1949. He improved only slightly, and on February 25, 1949, a subtotal colectomy was done. After this, he gained rapidly until March 11, 1949, when he was suddenly seized with severe pain in the left lower chest, accompanied by dyspnea, orthopnea and marked anxiety. Physical examination at this time revealed a cyanotic, extremely apprehensive man, with marked restriction of respiratory excursions. There was + pitting edema of the right lower leg, with calf tenderness and a positive dorsiflexion sign. Opera-

TABLE 1. *Summary of Statistics of 4 Cases Treated by Unilateral Procaine Stellate Block.*

CASE NO.	AGE	PRIMARY DISEASE	DURATION OF EMBOLUS BEFORE BLOCK	SYMPTOMS				RESULT OF BLOCK	POSTEMBOLIC PROPHYLACTIC MEASURES
				CHEST PAIN	DYSPNEA	CYANOSIS	SHOCK		
	yr.								
1	32	Ulcerative colitis	1 hr.	+	+	+	0	Complete relief	Bilateral superficial femoral vein ligation
2	41	Pulmonary tuberculosis	5 min.	+	+	+	+	Complete relief	Bilateral superficial femoral vein ligation
3	54	Arteriosclerotic heart disease	12 hr.	+	+	+	0	Complete relief of chest pain	Bilateral superficial femoral vein ligation
4	35	Postphlebitic syndrome	30 hr.	+	0	0	0	No effect	Inferior vena cava ligation

tion for bilateral superficial femoral-vein ligation was begun, under local anesthesia, within one hour after the acute onset of pain. A thrombus was removed from the right superficial femoral vein. During the operative procedure a left stellate-ganglion block produced complete relief of chest pain, respirations dropped from 40 to 18 per minute and dyspnea, orthopnea and cyanosis disappeared. The patient remained free of these symptoms and progressed to complete recovery.

Case 2. (CVAH, 11822). T. C., a 41-year-old man, was admitted to the hospital for the first time on September 13, 1948, for treatment of active pulmonary tuberculosis. X-ray examination of the chest on admission showed a cavity in the first left anterior intercostal area 2 cm. in diameter, with perifocal inflammation. In the lateral view this was seen to be in the apex of the left upper lobe. Pneumothorax, initiated shortly after admission, was unsuccessful because of adhesions. In view of the patient's failure to improve under conservative treatment, thoracoplasty was advised. On December 20, 1948, ten days after the second-stage thoracoplasty, the patient suddenly developed a severe pain in the right posterior chest, accompanied by dyspnea, orthopnea, cyanosis and slight hemoptysis. Physical examination revealed an apprehensive patient with marked splinting of the right chest and grunting respirations. In addition, tenderness was present on palpation of the right calf. Dorsiflexion of the foot caused no calf pain. A bilateral superficial femoral-vein ligation under local anesthesia was begun within two hours of the onset of the acute episode. No thrombus was found at the site of ligation. While the operation was in progress the patient complained of a sudden exacerbation of pain in his right chest. This was followed almost at once by vomiting, increased cyanosis, impairment in the quality of his pulse and absence of a demonstrable blood pressure. One hundred per cent oxygen was given, and a stellate block on the right side was immediately performed. Within five minutes the chest pain was gone completely, the pulse was of good quality and the blood pressure returned to normal levels. The respiratory rate, which had been 30 to 34 per minute since his arrival in the operating room, decreased to 18, and the patient voluntarily stated that he could breathe more easily. Postoperatively his convalescence was uneventful, and he had no recurrence of chest pain. He underwent a third-stage thoracoplasty several weeks later and eventually was discharged as asymptomatic, with a negative sputum.

Case 3. (CVAH, 13808). J. P., a 54-year-old man, was admitted to the hospital for the first time on January 5, 1949, with a chief complaint of progressive weakness and shortness of breath of five months' duration. Past history revealed a coronary thrombosis in 1945. Physical examination showed a chronically ill, apprehensive man with a blood pressure of 170/100. There was cyanosis of the lips and nails, moderate respiratory distress in the sitting position and distention of the neck veins. Numerous wheezes and rhonchi were heard over both lung bases. The heart was slightly enlarged. There was + pitting edema of the ankles. He was placed on a low-salt diet, digitalis and diuretics, but cardiac decompensation progressed slowly. On March 8, 1949, he complained of sudden pain in the left upper chest. Cyanosis and orthopnea increased markedly. Examination of the legs revealed increased edema of the right ankle and slight tenderness in the right calf. Dorsiflexion of the foot caused no discomfort. X-ray examination of the chest showed an area of density in the peripheral portion of the left upper lobe, indicative of pulmonary infarction. An electrocardiogram was also suggestive of this finding. A bilateral superficial femoral-vein ligation was performed under local anesthesia about twelve hours after the episode of pulmonary embolism. The veins were free of thrombi at the site of ligation. Because of the persistent chest pain, marked dyspnea, orthopnea and cyanosis, a left stellate-ganglion block was performed during the operation. The chest pain immediately subsided, but there was no decrease in the respiratory rate. Dyspnea and orthopnea were slightly diminished. After operation the patient improved for several days, without return of the chest pain, but died four days later after a cerebral vascular accident. Autopsy showed multiple pulmonary infarcts, coronary occlusion with a healed area of infarction, and recent infarction of the right frontal, parietal and temporal regions of the brain.

Case 4. (CVAH, 18434). T. K., a 35-year-old man, was readmitted to the hospital for the second time on August 29, 1949, because of ulcers on the right ankle of eight months' duration. The previous admission, in November, 1947, had been for treatment of thrombophlebitis of the right leg. He was given anticoagulants, having had a bilateral superficial femoral-vein ligation at another hospital two months previously. In January, 1949, six months prior to his present admission, he developed ulcers over the right lateral and medial malleoli. He sought hospitalization when ambulatory treatment failed. Physical examination was negative aside from bilateral longitudinal scars just below the groin and a small ulcer over each malleolus of the right ankle. It was believed that lumbar sympathectomy might be helpful in healing the ulcerations, and this operation was performed on the right side on September 14, 1949. The ulcers showed steady improvement, but on September 27, 1949, the patient coughed up blood-flecked sputum. On the next day he complained of right-chest pain, the blood-streaked sputum continued and his temperature and pulse were slightly elevated. On September 29, 1949, a chest film showed evidence of an infarction in the right lower lobe. Because of the persistent chest pain a right stellate-ganglion block was performed. No relief of the chest pain was obtained, nor were the pulse and respiratory rates altered, although the patient did feel that he was able to breathe more deeply. His inferior vena cava was ligated the same day. He was discharged asymptomatic on October 28, 1949.

Comment

Although such a small series of cases is not statistically significant, the findings suggest that the reflex spasm that occurs immediately after pulmonary embolism can be modified by stellate block. It is possible that the relief of spasm may diminish the area of lung that becomes infarcted. After an indeterminate period of about twelve hours, however, it has been our experience that stellate block is useless as relief, either because the spasm has subsided or because the effects of the occlusion have become too firmly established.

Without discussing the subject in detail, it should be pointed out that atropine may be used in the early treatment of pulmonary embolism to inhibit vagal impulses and papaverine may be used to relax smooth muscle. These drugs are undoubtedly beneficial when administered alone or in conjunction with other therapeutic measures.[9] The fact that papaverine has a widespread effect and does not prevent smooth muscle from responding to stimuli[19] would make its use seem less desirable than the more localized and complete effects of stellate block.[20]

It is possible that in carrying out the procaine block as described above some of the solution may diffuse beyond the stellate ganglion and involve an indeterminate number of the upper-thoracic sympathetic segments. If so, we feel that this would augment rather than diminish the relief in spasm in the pulmonary tree brought about by stellate block alone.

Summary

Four cases of pulmonary embolism treated by stellate-ganglion block are presented.

In the 3 cases in which stellate-ganglion block was carried out within a few hours of the acute episode symptoms were immediately relieved,

whereas in the 4th case, in which the block was delayed, little improvement was noted.

The possible explanation for the relief of symptoms is discussed.

References

1. Trendelenburg, F. Ueber die operative Behandlung der Embolie der Lungenarterie. *Arch. f. klin. Chir.* 86:686-700, 1908.
2. Churchill, E. D. Mechanism of death in massive pulmonary embolism, with comments on Trendelenburg operation. *Surg., Gynec. & Obst.* 59:513-517, 1934.
3. Gibbon, J. H., Jr., and Churchill, E. D. Physiology of massive pulmonary embolism: experimental study of changes produced by obstruction to flow of blood through pulmonary artery and its lobar branches. *Ann. Surg.* 104:811-822, 1936.
4. Nygaard, K. K. Pulmonary emboli; consideration of clinical diagnosis and possibilities for Trendelenburg operation. *Proc. Staff Meet., Mayo Clin.* 13:586-589, 1938.
5. Redon, H. Traitement chirurgical de l'embolie pulmonaire. *J. de chir.* 62:267-273, 1946.
6. Leriche, R., Fontaine, R., and Friedmann, L. L'infiltration stellaire est-elle justifiée dans l'embolie pulmonaire du point de vue physiologique et anatomo-pathologique? *J. de chir.* 50:737-748, 1937.
7. Bagenat, W. E., and Rapee, L. A. Treatment of pulmonary embolus by stellate block. *Anesthesiology* 8:500-505, 1947.
8. Neuhof, H. *Venous thrombosis and pulmonary embolism. Monograph No. 2, Mt. Sinai Hospital.* 159 pp. New York: Grune & Stratton, 1948. P. 147.
9. deTakats, Geza, et al. Pulmonary embolism, experimental and clinical study. *Surgery* 6:339-367, 1939.
10. deBurgh Daly, I. Physiology of the bronchial vascular system. *Harvey Lect. Ser.* 31:215-255, 1936.
11. deBurgh Daly, I., Ludány, G., Todd, A., and Verney, E. B. Sensory receptors in pulmonary vascular bed. *Quart. J. Exper. Physiol.* 27:123-146, 1937.
12. Mulvihill, D. A., and Harvey, S. C. Studies on collateral circulation. I. Thermic changes after arterial ligation and ganglionectomy. *J. Clin. Invest.* 10:423-429, 1931.
13. Dack, S., Master, A. M., Horn, H., Grishman, A., and Field, L. E. Acute coronary insufficiency due to pulmonary embolism. Electrocardiogram and pulmonary embolism. *Am. J. Med.* 7:464-477, 1949.
14. White, J. C., and Smithwick, R. H. *The Autonomic Nervous System.* 469 pp. New York: The Macmillan Co., 1941.
15. Carr, D., and Chandler, H. Dorsal sympathetic ganglionectomy for intractable asthma. *J. Thoracic Surg.* 17:1-12, 1948.
16. Kirgis, H. D., and Reed, A. F. Temporary interruption of sympathetic impulses to head by infiltration of cervical sympathetic trunk. *Ann. Surg.* 128:101-109, 1948.
17. deSousa Pereira, A. Blocking of middle cervical and stellate ganglions with descending infiltration anesthesia: technic accidents and therapeutic indications. *Arch. Surg.* 50:152-165, 1945.
18. Caldwell, G. A., Broderick, T. F., Jr., and Rose, R. M. Sympathetic block of stellate ganglion. *J. Bone & Joint Surg.* 28:513-520, 1946.
19. Goodman, L., and Gilman, A. *The Pharmacological Basis of Therapeutics.* 1383 pp. New York: The Macmillan Co., 1941.
20. Crowley, L. G., Murphy, R. A., Jr., and Cooper, F. W., Jr. Release of vasoconstriction by vasodilator drugs and sympathetic nerve block. *Surgery* 27:879-887, 1950.

REVERSIBLE UREMIA WITH HYPERCALCEMIA DUE TO VITAMIN-D INTOXICATION*

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CHRONIC renal disease with uremia is usually an irreversible process. A case of reversible uremia is therefore reported as a reminder that the diagnosis is simple if the cause is present and is thought of but is often overlooked because its possibility is not considered. If it is recognized early, cure can be expected.

Case Report

A 40-year-old advertising executive was first seen on February 28, 1950, because of progressive weakness, fatigability, dryness of skin and mouth, excessive thirst and polyuria. His past history was irrelevant except for the following: At the age of 14, during a routine insurance examination, albumin was found in his urine. For the three subsequent years, with a diagnosis of nephritis, he was under the care of an able internist who kept him on a low-protein, low-salt diet. At the end of three years the situation was regarded as cleared. He was able to obtain life insurance, although there was a slight mark-up of the premium rate. He remained well until the age of 20, when albumin was again discovered at an insurance examination. For the two subsequent years he was under the care of the same physician, again on restricted salt and protein.

After presumable recovery from this episode, he remained perfectly well until the onset of his present illness. He was able to obtain several more life-insurance policies at normal premium rates and was even able to have the mark-up on the first policy removed. His systolic blood pressure was never over 120; the diastolic pressure was not over 90, except during the two attacks of nephritis, when he thought the readings were somewhat higher.

In November, 1949, at the age of 40, four months prior to our first examination, he developed what he and his

Table 1. *Urine.*

Date	Fluid Balance			Albumin	Specific Gravity	Phenolsulfonphthalein Excretion					Calcium Excretion (Sulkowitch)
	Day	Night	Total		Highest concentration	15 min.	30 min.	1 hr.	2 hr.	Total	
	cc.	*cc.*	*cc.*			%	%	%	%	%	
March 1-5 ..... (average)	In 4120 Out 2910	In 700 Out 1890	In 4820 Out 4800	0-1+	1.010	15	5	10	10	40	3+
April 26	In 1600 Out 1140	In 360 Out 780	In 1960 Out 1920	1+	1.009	6	8	7	3	24	—
September 29...	In 1320 Out 1170	In 900 Out 990	In 2220 Out 2160	1+	1.018	23	10	8	6	47	Normal

Table 2. *Blood.*

Dates	Hemoglobin	White-Cell Count	Blood Film	Serum Nonprotein Nitrogen	Serum Calcium	Serum Potassium
	gm./100 cc.	*x10³*		*mg./100 cc.*	*mg./100 cc.*	*milliequiv./liter*
March 1-5	9	7.5	Normocytic mild hyperchromic anemia	50	14.1	4.8
April 26	10.3	8.6	Same	33	10.1	—
September 29	12.5	9.0	Normal	25	9.8	—

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physician thought was typical grippe. He had a running nose, cough and bone aches; his temperature rose to 100°F., but no higher. He was treated with penicillin and was absent from work for only one week. However, after returning to his office in mid-November, he did not feel entirely well. The first symptom was persistent fatigue. He could accomplish very little work in the afternoon, and arrived home each evening so exhausted that he found it necessary to retire immediately. During this same period, he occasionally felt a little chilly, as if he might have a slight fever; he noticed some stuffiness of the nose, which could be controlled by one or two aspirin tablets. These latter symptoms alone were never sufficient to interfere with work; fatigue was the predominant problem. He also developed some trouble in controlling his urine — that is, when his bladder became moderately full he would have to void immediately or lose control. There was no nocturia. When he stopped drinking his customary 6-8 daily cups of coffee, the urgency promptly ceased. Because withdrawal of the coffee appeared to cause constipation he resumed his previous intake, and the urgency reappeared. Meanwhile, in mid-January, about six weeks prior to his first visit, he developed persistent unquenchable thirst and resultant polyuria. His fluid intake rose to more than a gallon a day and he voided large amounts approximately every two hours, day and night. There were no other urinary symptoms. His skin and mouth became dry; distressing generalized pruritus set in. There was no appreciable loss of weight or other symptoms except greater fatigability.

The patient looked worn and pale. The skin and mucous membranes were dry. The blood pressure was 140/80. There were no other pertinent abnormal physical signs. His prostate was normal in size; its secretion showed no evidence of infection. A single specimen of urine passed in the office was normal except for a specific gravity of 1.008. Examination of the blood revealed a hemoglobin of 9 gm., a red-cell count of 3,460,000 and a white-cell count of 10,300, with a normal differential count. The stained film showed evidence of normocytic, slightly hypochromic anemia. A blood Hinton test was negative. The nonprotein nitrogen was 61 mg. per cent.

The patient was promptly admitted to the Massachusetts General Hospital for further investigation. Initially it was thought that he had either chronic glomerular nephritis, hyperparathyroidism or perhaps pure diabetes insipidus, although it was recognized that the last-named would not account for the elevated nonprotein nitrogen and the anemia. A decided source of confusion was the normal blood pressure in the presence of what appeared to be severe chronic renal impairment.

On admission, physical examination was unchanged except for a blood pressure reading of 160/90. Typical band keratitis was later found in both eyes.

The results of pertinent laboratory studies are noted in Tables 1 and 2.

The glucose tolerance test showed a mild diabetic curve; a bromsulfalein test showed only 2 per cent retention of dye. No evidence of intrinsic bone change was found on x-ray films of skull, spine and pelvis. The lamina dura around all teeth was intact. No trace of calcium deposit in the region of the kidneys was seen in a plain film of the abdomen. An electrocardiogram was normal except for a short QT interval, which is reported to be common in hypercalcemia.

Because the case obviously did not conform to the pattern of any of the usual forms of renal disease or to hyperparathyroidism or pituitary diabetes insipidus, and because the diagnosis appeared obscure, Dr. George W. Thorn was consulted. In addition to the diagnoses mentioned above, he raised the question of three other possibilities: chronic pyelonephritis with hypercalcemia and alkalosis, as described by Burnett and his co-workers[1]; metastatic disease such as myeloma; vitamin-D intoxication superimposed on chronic nephritis.

Further questioning of the patient brought out the fact that he had been taking 200,000 units of vitamin D daily for approximately six months. It had been prescribed by another physician for pain in the shoulder. The diagnosis was thus established.

After six days of hospitalization, the patient was discharged on a low-calcium diet and a regimen of forced fluids. During the subsequent seven months he has shown continued clinical improvement. On September 29, 1950, he reported that he felt very well and that since June he had been able to perform a full day's work without difficulty. Fatigue, thirst, polyuria and all other symptoms had entirely disappeared. His blood pressure was 120/70. The band keratitis had also cleared. This observation was confirmed by an ophthalmologic consultant.

The progress of pertinent laboratory findings is shown in Tables 1 and 2.

The manifestations of vitamin-D intoxication, as recently summarized by Chaplin, Clark and Ropes, are indicated below.[2] Not all these are necessarily present in a single case.

General: weakness, fatigue, weight loss.

Gastrointestinal: nausea, vomiting, diarrhea, abdominal cramps.

Neuropsychiatric: headache, paresthesia, vertigo, variable mental symptoms (depression, mild psychosis, coma).

Hematologic: normocytic, normochromic anemia.

Urologic: red cells, casts, albumin in the urine, with progressive impairment in concentrating ability and symptomatic frequency and nocturia.

Chemical: elevated serum calcium and phosphorus, with normal or slightly elevated alkaline phosphatase; progressive nitrogen retention.

Roentgenologic: diffuse demineralization of the bones in advanced cases; periarticular soft-tissue calcifications.

Pathologic: scattered calcium deposition in heart, kidney, blood vessels, stomach, lung, thyroid, pancreas and periarticular soft tissues.

Ophthalmologic: band keratitis.

The case reported above showed weakness and fatigue, band keratitis, mild anemia that was slightly hypochromic as opposed to the normochromic anemia usually reported, polyuria, impairment of urine concentration, slight albuminuria without abnormal solid elements, nitrogen retention and elevated blood calcium. Blood phosphorus was normal in contrast to the slightly elevated reading usually reported. The normal blood phosphorus and phosphatase and the absence of

TABLE 2 (*Concluded*).

DATES	SERUM PHOSPHORUS (INORGANIC)	SERUM ALKALINE PHOSPHATASE	SERUM SODIUM	SERUM CHLORIDE	SERUM CARBON DIOXIDE CONTENT	SERUM CHOLESTEROL	TOTAL SERUM PROTEIN
	mg. /100 cc.	*units /100 cc.*	*milliequiv. /liter*	*milliequiv. /liter*	*milliequiv. /liter*	*mg. /100 cc.*	*gm. /100 cc.*
March 1-5	5.0	3.	147	100	33	198	7.5
April 26	4.0	3.6	—	—	—	—	—
September 29	5.3	7.8	139	97	31.8	—	—

any bone changes at x-ray examination tended to exclude hyperparathyroidism. The result of simple therapy, notably cessation of the vitamin-D intake, low calcium intake and increased fluids, is evident from the protocol.

Summary

A case of vitamin-D intoxication is reported. The outstanding features were increased fatigue and weakness, polydipsia and polyuria, nitrogen retention and a high blood calcium with relatively normal levels of other chemical elements. Improvement since the omission of vitamin D has been most satisfactory. This case is reported in order to emphasize the danger of large doses of vitamin D and the necessity of considering overdosage of this vitamin as a possible cause of uremia and hypercalcemia.

References

1. Burnett, C. H., Commons, R. R., Albright, F., and Howard, J. E. Hypercalcemia without hypercalcuria or hypophosphatemia, calcinosis and renal insufficiency: syndrome following prolonged intake of milk and alkali. *New Eng. J. Med.* 240:787-794, 1949.
2. Chaplin, H., Jr., Clark, L. D., and Ropes, M. W. Hypervitaminosis-D. *Am. J. M. Sc.* (in press).

FIFTY YEARS OF MEDICAL PROGRESS

MEDICINE AS A SOCIAL INSTRUMENT: MILITARY MEDICINE

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THE twentieth century has been an era of far-reaching extension of the frontiers of medicine. In this, military medicine has had a conspicuous part. The range from the very modest resources and knowledge with which the medical responsibilities of the Spanish-American War were met to the vast global medical enterprises of World War II exemplifies the progress of medicine in general. Military medicine is such a complex of skills and so closely interwoven with the total practice of medicine that any effort at concise evaluation of the influence of military and civilian medicine on each other is likely to become tedious indeed. Some signal accomplishments have been totally military; others have resulted entirely from the necessities of war when a vast military organization challenged civilian doctors and scientists in uniform to hasten medical progress and bring to a rapid conclusion researches, developments and practices that might otherwise have been slow of evolution.

No effort will be made here to differentiate these accomplishments. The purpose of this paper is to record those outstanding medical developments for which the Military Medical Service has been responsible or which it has materially influenced. For the most part, the details of individual contributions must be left to the historian and to those who wish to review the voluminous literature of the period.

The primary mission of the Military Medical Service is to conserve military manpower. For this purpose the Medical Service is dedicated to assembling, developing and applying knowledge that will maintain health in large groups. This implies selection and physical and mental classification of personnel, promotion of physical and mental well-being, prevention of disease and injury, treatment of the sick and injured and rehabilitation of the disabled. Furthermore, in both peace and war it implies preparation for these responsibilities through carefully planned and executed research programs and orderly progress in administration, organization, personnel management and medical logistics.

Although the basic concepts of health preservation do not vary between military and civil populations, operations, methods, administration and organizational procedures may differ greatly. Military populations are selected according to definite standards of physical and mental fitness and by age groups; for the most part, in the first half of the present century, they were limited to the male sex. Movement characterizes their missions. By necessity they live as large groups, are sometimes exposed to the extremes of climate, often endure unusual stresses and frequently must live and operate in "unsanitated" surroundings. These conditions favor the spread of disease, increase emotional stress and complicate the application of accepted preventive and clinical medical practice. Expeditious prevention and treatment of disease and injury from these circumstances require qualified military surgeons whose specialized training allows them to practice an exacting profession while at the same time promoting efficient performance of the military force. Additionally, in modern war, the Armed Forces are charged with the maintenance of practical health levels in civilian populations in liberated or occupied territories and with the proper care of prisoners of war in accord with international law.

The progress of events that led to this broad view of military medical responsibility was an evolution, most of which took place in the first half of the

twentieth century. World Wars I and II were the high points in the process.

Organization and Administration

Prior to and during the Spanish-American War the Medical Department consisted only of physicians and the enlisted men of the Hospital Corps. There was no permanent nurse corps, although volunteer female nurses served under contract. Dental service was not provided for the soldier; veterinarians served variously as enlisted men and civilian employees of the Army. Administratively, the Medical Department did not participate in military plans and decisions; rather, its personnel were regarded as an attached organization to care for the sick and wounded of the Army without representation in its councils. Stockage of reserve medical supplies for emergency use was not permitted, which made provision of medical service extremely difficult during the war. The Dodge Commission, appointed to investigate the conduct of the war, commented both critically and favorably on the medical service and found that causes for many of its failures lay outside the Medical Department.

In the decade following the war Congress increased the size of the Medical Department, gave statutory recognition to the Nurse Corps, created the General Staff and authorized the Medical Reserve Corps. There was greatly increased participation by Medical Department personnel in Army training and military activities. Entrance of the United States into World War I found the Medical Department better prepared to meet its responsibilities than at any time in its earlier history. The responsibilities were also greater than ever before; the Army increased from 100,000 to 4,000,000. In 1918 there were over 2,400,000 admissions to sick report owing to disease alone.

To meet the problem of providing medical service to the wartime Army, Surgeon-General William C. Gorgas expanded his office from 5 small to 20 large divisions, embracing not only the well established activities of medical practice, sanitation and administration but also new fields such as psychology, roentgenology, nutrition and gas warfare. On entrance into the war the Medical Department numbered 981 officers, 403 nurses and 6619 enlisted men. During the war its total personnel was expanded to almost 30 times the initial strength. When the Armistice was signed, there were 91 military hospitals in the United States, with over 120,000 beds.

Training for large numbers of medical officers and enlisted technicians was provided by the Surgeon General in numerous training centers and to a smaller extent at civilian institutions. The newly created National Research Council and the American Medical and Dental Associations gave assistance in medical research, personnel procurement and technical medical problems. The American Red Cross recruited nurses and furnished ambulance services and medical supplies, both at home and in France. Problems relating to the determination of requirements, procurement of supplies and transportation of thousands of personnel and millions of tons of material over long distances brought about a new specialty in military science — logistics.

In France, General Merritte W. Ireland, after considerable difficulty, established the Medical Service as an integrated part of the military organization and effort. As Surgeon of the American Expeditionary Forces, he had offices both in the Services of Supply and in General Headquarters, where he received complete support of the American Commander in co-ordinating the Medical Service with administrative, supply and tactical operations.

The Dental Corps and the Veterinary Corps became component parts of the Medical Department in 1911 and 1916 respectively; during World War I the Sanitary Corps was formed to provide an organization in which sanitary engineers, psychologists and other experts in fields related to medicine could be commissioned. Physicians qualified in the various medical and surgical specialties were brought into the Office of the Surgeon General to advise on matters pertaining to their specialized fields, and eminent surgeons and internists functioned as consultants in the American Expeditionary Forces.

World War I brought full recognition of the magnitude and complexity of the medical service necessary for large armies fighting overseas in modern war. Training, personnel administration, hospital construction and operation, professional specialization and integration with other activities of the Army were accepted as fundamental to the growth and full use of the Medical Service. After the war a Medical Field Service School was organized to train newly commissioned officers of the Medical Department in the essentials of military medical service, logistics and sanitation. The Army Medical School, organized before the beginning of the century, continued to teach young medical officers those aspects of medicine related to Army service that were not taught or sufficiently emphasized in medical schools. Reserve components of the Medical Department were built up, active and inactive training in which was an important factor of preparedness for World War II. Reserves of medical supplies were assembled and tables of organization for field medical units were prepared. The Surgeon General continued to operate as a bureau chief in the War Department, with considerable freedom of action.

The national emergency in 1939, followed by entrance into war in 1941, brought to the Medical Department many of the same problems faced in 1917, but on a larger scale. The Army at peak strength numbered over 8,000,000, as contrasted to 4,000,000 in World War I, and fought in many parts of the world. The war lasted four years in-

stead of one and a half years. As in 1917 and 1918, professional associations, the National Research Council and other governmental agencies assisted in the solution of technical and professional problems. Co-ordination, joint administration and technical advances made possible the welding of an effective program in a short time.

Many specialists in the ancillary medical sciences were commissioned in the Sanitary Corps, an organization that was retained as a reserve component between World War I and World War II. The Medical Administrative Corps, created after World War I, provided for the commissioning of officers in medical supply and other administrative activities.

After the war, provision was made for including men in all specialties essential to complete medical service as commissioned officers in the Medical Department (renamed Medical Service in 1950). The Sanitary Corps and Medical Administrative Corps were replaced by the Medical Service Corps, in which sanitary engineers, nutritionists, biochemists, bacteriologists, psychologists, other scientists, administrators and pharmacists were commissioned. The Women's Medical Specialist Corps was created to include sections for physiotherapists, dietitians and occupational therapists. An intensive program of postgraduate and residency training was instituted to build a medical service of fully competent professional officers. During the last five months of 1950, after hostilities had broken out in Korea, this program was already paying dividends in reducing disability and loss of American lives.

Preventive Medicine

The beginning of the twentieth century witnessed the inception of military preventive medicine. It grew out of the vigorous new science of bacteriology and the unfortunate experiences with communicable diseases during the Spanish-American War. Largely unorganized at first, it progressed under the leadership of the Army Medical School, through the Tropical Medical Research Board and the efforts of a group of talented officers who had embraced bacteriology and the field-and-laboratory-research method. George M. Sternberg of the Army and William C. Welch of the Johns Hopkins School of Medicine were the preceptors. Fundamental studies on insect-borne diseases, typhoid fever and other gastrointestinal diseases, nutritional deficiencies and sanitation followed.

Functional organization of military preventive resources first evolved in World War I. Epidemiology, the laboratory sciences and sanitary practice were focused upon the control of the respiratory, acute infectious and gastrointestinal diseases and the destruction of lice. The epidemiologic-laboratory team for the expeditious survey and focal attack of disease outbreaks was the signal preventive-medicine development of the war. For the first time in a theater of war, American combat forces had fewer deaths from disease than from battle injury.

Preventive medicine and public health had matured by the outbreak of World War II. Their outstanding contribution was the forging of a versatile preventive-medicine service, centrally and by echelon in the field, that brought together in coordinated effort the many scientific disciplines essential to health protection in a global conflict. Supporting this was the Army Epidemiological Board, composed of prominent civilian experts in preventive, laboratory and clinical medicine. As consultants, the members of the board and its commissions have extended the boundaries of military and civilian medicine by research and investigation of important disease problems at home and overseas. As a tri-service organization at the half-century, it stands as a bulwark of preventive-medicine practice and research.

Preventive medicine and public-health supervision had come to be recognized as major medical specialties for which the demonstration of health maintenance and disease control in World War II was in no small way responsible.

Insect-borne diseases. Insects are vectors of many diseases of great military importance. Much of the American military experience early in the twentieth century was in the tropics, where these diseases abound. Ross's discovery of the anopheline mosquito as the vector of malaria opened a totally unexplored field, which military medicine was strategically situated to foster. General Sternberg recognized this, and by encouraging the efforts of Major Walter Reed he set in motion a chain reaction of advances in tropical medicine in which military medicine was long pre-eminent. Reed's bold investigations on yellow fever and his discovery of the *Aedes aegypti* mosquito as the vector set the stage for the practical eradication of the urban disease, first demonstrated by Major William C. Gorgas in Havana. Similar environment and vector control under Gorgas in Panama limited malaria and yellow fever and made possible the construction of the Panama Canal. Service studies revealed a heretofore unknown malarial parasite, emphasized the importance of human inapparent cases in the spread of malaria and clarified the etiology and mosquito transmission of dengue fever.

Control of breeding areas by drainage, oil and Paris green as antilarval measures, adult mosquito destruction, protection of personnel by appropriate clothing, screening and bed nets and treatment of cases with quinine and later with atabrine constituted malaria control up to the time of World War II. Suppressive therapy with quinine, as applied, was not highly successful. Pyrethrum, limited in production to East Africa, was the only insecticide of military merit.

Epidemic typhus fever has traditionally accompanied wars, but adequate control of the ubiquitous louse had eluded combat forces. The cumbersome, expensive delousing stations of World War I failed because disinfestation of personnel and clothing gave no protection against immediate reinfestation.

Developments in World War II provided effective tools for the control of typhus fever and malaria. DDT, formulated in Switzerland and developed in the United States by the Armed Forces, the Department of Agriculture, the National Research Council and industry, was dramatically effective in control of the louse. When it was dusted into the hair, clothing and bedding, its lasting residual lethality to the louse made possible louse-free armies and the prevention of spread of typhus in civilian populations. The latter was conclusively demonstrated in the Naples outbreak and later in Western Europe.

In combat areas DDT as an areal antilarval agent, and as an aerosol and residual spray against adult mosquitoes, added greatly to the malaria-control armamentarium. Residual spraying in stable communities has brought within range the elimination of malaria as a public-health problem in entire countries. Fewer than 10 primary cases were reported in the United States in the fiscal year 1950, and dramatic results have been achieved in Venezuela and British Guiana. Mosquitoes, lice, sand flies and fleas, all vectors of militarily important diseases, and a variety of pests and agricultural insects can be controlled with DDT. Evolution of resistant strains of flies and other insects adds emphasis to further insecticidal research.

The World War II malaria-control and survey teams, embodying the skills of preventive medicine, entomology and sanitary engineering, emerged as a fundamental resource in the control of insect-borne disease.

Other communicable diseases. Seasoning, or herd resistance, has lowered the susceptibility of the American adult population to infectious diseases. In part, urbanization, rapid transport and population movements incident to the two great wars brought this about. Seasoning, improved immunization, the sulfa drugs and antibiotics have permitted a shift of emphasis from the great concern with measles, meningitis, mumps, diphtheria, pertussis and mortality from complicating pneumonia to other problems, including noncommunicable diseases and trauma.

Typhoid-paratyphoid vaccine, first used experimentally in the German and British Armies, was modified by Major F. F. Russell and has been mandatory for United States Forces since 1911. Typhoid fever has been of little military moment since, although parallel improvement in sanitation and control of food and water had their effect, as demonstrated by the lower incidence of other gastrointestinal diseases. Prior to World War II, prophylactic tetanus antitoxin, administered to the injured, was the mainstay of tetanus control. Administration of the toxoid for immunization of the Armed Forces in World War II was a vast field test that conclusively demonstrated the procedure. Twelve cases occurred; 6 were not fully immunized and 5 died. This was a fatality rate of 0.44, as compared with 13.4 in World War I and 2.4 between the wars. Segments of American forces received yellow-fever and typhus-fever vaccines but, as in the case of typhoid-paratyphoid vaccination, mass exposure to the analogous disease was not sufficient for evaluation. Cholera and plague vaccines were shown to give partial protection, while the mid-century saw vaccines against Japanese B. encephalitis, influenza, mumps and dengue fever under study. Vaccines for bacterial pneumonia, the dysenteries, gas gangrene and scrub typhus have not yet met with success. Immunization in the controlled population of the Armed Forces has tested biologicals on a vast scale, advanced the technics of vaccine production and fostered high standards of assay, purity and protection.

The long sought control of communicable disease by chemical prophylaxis made little progress before 1940, except for limited success with quinine against malaria. In the absence of quinine supplies, atabrine was effective in the suppressive treatment of malaria, once the dosage schedule had been determined, and undoubtedly was better tolerated than quinine. Chloroquin, one of 14,000 compounds evaluated, is the current drug of choice. Others developed since the war show much promise; none are true causal prophylactics. Sulfadiazine simplified the control and treatment of meningitis. No large outbreaks occurred, and the mortality was reduced from 38 per cent in World War I to less than 5 per cent in World War II. Experimental chemical or antibiotic prophylaxis in the streptococcal infections and for the dysenteries was determined to have promise. Chloramphenicol is of potential military importance for the suppression of scrub typhus.

Field sanitation. Sanitation for temporary areas occupied by large groups is a perennial military problem. Reed, Vaughan and Shakespeare concluded in part that typhoid fever in the Spanish–American War was the result of pollution of the camps and that flies were carriers of the infection. By the end of 1950, flies still resisted complete control and maintenance of cleanliness remained subject to the human element. Disposal technics for excreta and other wastes were greatly improved early in the century. Little has been added to the principles of field practice, although disinfectants and experimental activators have expedited waste sterilization. Disposal systems, patterned after municipal systems, were installed in camps and stations. Incineration and the sanitary-fill technic safely disposed of garbage. Practical water purification originated with the development of a filter

and the adaptation of liquid chlorine and chlorine compounds to field use by Major Carl R. Darnall. Water treatment with chlorine or chlorine compounds prevailed through World War I and II and became a standard procedure in municipal water plants in many parts of the world. Small field equipment embodying the principles of sedimentation, chemical flocculation and settlement, filtration and chlorination supplied field units. Calcium hypochlorite and Halazone tablets provided potable water for the individual soldier. Two late developments, the diatomaceous-earth filter, field-tested toward the end of World War II, and Globaline, an iodine preparation, give promise of protection against amebic dysentery as well as the intestinal diseases of bacterial origin.

Sanitary science has developed greatly by virtue of the necessities of military sanitation and the high standards of water purification, waste disposal, insect control, food inspection and mess sanitation maintained in the military forces.

Nutrition and management of food. In the first decade of the century, Captain E. B. Vedder demonstrated in the Philippines that beri-beri is a nutritional deficiency disease — a field that was enriched by Colonel Joseph Siler's studies of pellagra in the United States. More important for the Armed Forces were the studies of normal nutritional requirements and improvements in food procurement, storage and distribution made during the half-century. There were no "embalmed beef" scandals after 1900. The science of nutrition, applied jointly by the Medical Department and the Quartermaster Department, provided packaged rations for World War II that were nutritious, safe and transportable, and without which the rapid movement tactics so characteristic of American arms would have been impossible. Of fundamental importance for the Armed Forces and for food standards in general was the vast program of food inspection executed by the Veterinary Corps. In addition, careful investigation of malnutrition and nutritional deficiencies in European populations that had long subsisted on ma.ginal diets substantially enhanced the knowledge of food utilization, protein deficiency and the pathologic physiology resulting from lack of enough calories and essential nutrients.

Prevention of trauma. Pirogoff in 1878 said that war is the epidemiology of trauma. A study of the causative factors in accidents was proposed in the Army in the 1930's; wound ballistics was investigated before World War I. However, it was not until World War II that accidents and trauma from cold injury were subjected to analysis to determine the relative weights of causative factors and their epidemiologic significance. Trauma is the preponderant cause of death in modern war. Little is known of the total pathogenesis of traumatism or of its intricate relations to liver function, the cardiovascular and hematopoietic systems, tissue fluids and the adrenocortical and other endocrine systems. Psychic stress and emotional immaturity are intimately related to accident proneness. Psychologic and physiologic responses to injury and stress require intensive study. Evaluation of these factors through research with irradiated metabolites and by other means, psychiatric investigation, and epidemiologic appraisal of total causation may greatly influence the development of principles and practices for the reduction of loss from traumatic injury both in military forces and in civilian life in the home, on the highway and in industry. The problem comes more dramatically into focus when the question of civil defense is entertained.

Professional Service

At the beginning of this century the army doctor served as physician, surgeon, public-health officer and administrator, a circumstance not greatly different from that of the physician in civilian life. Fifty years of medical advances have witnessed progressive specialization in medical practice and emphasis on medical teamwork by the physician, the dentist and the specialists in the sciences allied to medicine.

Progress in the medical sciences was translated into improved military medical practice in both wars. The integration of civilian practices into war medicine was greatly facilitated by the consultant system, through which eminent civilian medical specialists encouraged high professional standards, supervised medical care and detected and counseled on new clinical problems. Working as a team with Regular Army medical officers skilled in matters peculiar to military organization, administration and logistics, they established the system in World War I; in World War II they so thoroughly tested and demonstrated its merit that it has become a permanent feature of the Army Medical Service.

Surgery

Since the earliest times, military surgeons have been faced with the problems of applying whatever surgical technics were then in vogue to the treatment of wounds under the difficult conditions inherent in armed warfare, of controlling the inevitable infection of wounds, of preventing death from the condition that we now know as shock and of reconstructing the disabled or disfigured soldier into a happy and useful member of society. In 1900 relatively little had been accomplished in the solution of these problems, although better field medical service, developed by army surgeon Jonathan Letterman in the Civil War, did much to prevent loss of life among the wounded from exposure and lack of prompt medical attention. In the Spanish–American War the main problems were medical, not surgical; thousands of soldiers were sick and many had died, largely from diseases we now know to be preventable. It is not remark-

able that for the next eighteen years the major professional efforts of the Medical Department were directed toward problems of communicable disease control.

World War I was characterized by the development of tremendous fire power incident to the use of high-velocity bullets, automatic weapons, high-explosive shells, shrapnel and grenades. Heavy combat resulted in hundreds of cases of wounds that were often extensive and often accompanied by destruction of tissue. Multiple wounds were not infrequent. Surgical treatment of a high order, applied early if lives were to be saved, was needed in great quantity. The need was met by the combined efforts of a small nucleus of Regular Army medical officers and by the thousands of civilian physicians and dentists in the service, with the guidance of eminent surgical consultants. Mass surgery was practiced on a scale never before seen in American medicine. Traumatisms, rarely seen in civilian practice, were commonplace. It was not remarkable that medical experience in this war brought about marked improvement in surgical technics. Great progress was made in the fields of orthopedic surgery, neurosurgery, thoracic and abdominal surgery and plastic surgery. The importance of type of anesthesia in relation to type and extent of surgery was recognized, and improvements in anesthesia technics followed.

The need for early surgical aid was met by improving the means of evacuating the wounded and by moving surgical teams with their necessary equipment closer to the front-line casualty area in order to shorten the time between wounding and treatment. Motor vehicles were used for the first time in war; this made rapid evacuation possible.

A partial solution to the problem of wound infection was found. Prevention was not successful, but infection undoubtedly was deterred by local application of alcohol and tincture of iodine, together with the extensive use of sterile dressings. Wound infections were treated hopefully with Carrel–Dakin solution, a long and tedious process much esteemed during the war but later accepted with reservations in civil practice. The almost certain infection of wounds brought about the concept of late secondary closure. Complications from tetanus were few because of the general use of the antiserum; approximately 70 cases of tetanus were reported. Gas gangrene was not effectively controlled. However, the importance of devitalized tissue in the development of this complication was understood, and the concept of wound débridement was thoroughly accepted as an essential part of early surgery. Shock was recognized as a complication of severe wounds, and some progress was made in determining its nature and treatment. Shock wards, where patients were kept quiet and warm, were established in forward medical installations. Some use was made of saline infusions and blood transfusions. After the war much reconstructive and plastic repair was done, with remarkably good results.

Between the wars, military surgery progressed in general and station hospitals; it was greatly influenced by improvements in surgical technic in civilian practice. It was anticipated that two developments would profoundly influence military surgery. First, blood plasma was successfully dried in a form that made preservation, transportation and reconstitution relatively simple. Continued experimental research on shock in animals led many to conclude that infusions of reconstituted blood plasma would prevent and cure wound shock. Secondly, shortly before World War II various sulfonamide compounds were found to be efficacious in treatment of certain bacterial infections and were adopted in the belief that wound infections might be prevented by self-administered sulfonamide tablets taken orally by the wounded soldier and by sulfanilamide powder directly applied into the wound by aid men on the battlefield. The results were disappointing.

Organizationally, provisions were made for added mobile evacuation hospitals and mobile auxiliary surgical units designed to bring surgical treatment to the wounded soldier near the field of battle.

World War II brought few startling innovations in surgical technic. One of the most important surgical advances was the development of a rational concept of wound management that insured continuity in surgical procedures from the time of wounding to the complete repair of the resultant damage. While the sulfonamides did not evolve as a panacea for wound infection, the discovery and use of penicillin, together with improved surgical technics, resulted in the virtual elimination of this ancient foe of the military surgeon. Reconstituted blood plasma for shock failed of its early promise but filled a fundamental role as the parenteral fluid of choice for preliminary support and resuscitation on the battlefield and in other locations where blood transfusion was impractical. Whole-blood transfusion for adequate treatment of shock was proved effective early in the war. Continued study of wound shock in the field contributed much toward its understanding and successful prevention and treatment.

Other important developments in military surgery were:

Introduction of the concept of reparative surgery.

Provision of treatment facilities for the nontransportable wounded in the division area.

Recognition that primary operation for the wound is in itself a major agent of resuscitation.

Elimination of intrapleural infection and preservation of lung function in wounds of the chest.

Advances in maxillofacial and oral surgery and the development of tantalum and vitallium for the replacement of cranial bone, largely by dental specialists.

Development of the plastic artificial eye by dental personnel.

Acceptance of the concept of early secondary repair of most wounds, and primary closure of selected wounds.

Whole-blood transfusion for the traumatized is so important that the postwar period has seen great emphasis placed upon the development of blood banks and the technics of collection, storage and shipment of blood not only for military purposes but also for civil defense.

Thus, in the last fifty years, modern military surgery has found solutions to many of its problems. Evidence of the degree of success is found in the reduction of the case fatality rate in wounded men received in medical installations from 8 per cent in World War I to 4½ per cent in World War II, as shown in Table 1. This is highly significant in the light of the increasing number of severely wounded men brought to the surgeon for treatment by a constantly improving field medical service.

TABLE 1.* *Percentage of U. S. Army Wounded† Who Subsequently Died as a Result of Their Wounds.*

WAR	TIME PERIOD	PER CENT
Spanish-American	May 1898–December 1898	6.7
Philippine Insurrection	January 1899–December 1902	7.8
World War I	April 1917–December 1918	6.1 including gas casualties 8.1 excluding gas casualties
World War II	December 1941–August 1945	4.5

*From *Morbidity and Mortality, 1819-1944, U. S. Army. Weekly Health Rep. Vol. V* (No. 48), November 30, 1945. (Extract.)

†Wounded or injured in action; includes deaths from wounds but does not include deaths in action or deaths while prisoner of war.

Department of the Army
Office of the Surgeon General
Medical Statistics Division
12 January 1951

INTERNAL MEDICINE

Clinical medicine in the Armed Forces has not been merely the screening process of the sick call, but rather good general practice in the outpatient dispensaries and on small posts, and medicine in the hospitals that was neither better nor worse than contemporary civilian practice. Early in the period, emphasis was placed largely upon the care of the infectious, respiratory and gastrointestinal diseases. Studies in the Philippines demonstrated the therapeutic effectiveness of intravenous saline solution in the treatment of cholera, while Vedder established ipecac and later the emetine component of ipecac as the drug of choice in the treatment of amebiasis. World War I saw no dramatic developments in the management of disease. Treatment of many thousands of cases of acute infectious diseases, influenza and other respiratory diseases and gastrointestinal disease resulted in much improvement in medical standards. Opportunities were afforded for the study of unfamiliar diseases, such as trench fever, encephalitis lethargica, war nephrosis, neurocirculatory asthenia, trench foot and an array of mental-emotional aberrations commonly referred to as shell shock.

As in civilian medicine, the advent of the sulfa drugs revised much of accepted practice. They, together with penicillin, most profoundly influenced clinical medicine in World War II. Mortality and noneffectiveness from a number of diseases were drastically reduced. The venereal diseases, lobar pneumonia, meningitis and the dysenteries are outstanding examples. Penicillin cured gonorrhea in twenty-four to forty-eight hours on a duty status and inactivated syphilis in eight days. The corresponding saving in time, medical care and noneffectiveness were of essential military importance.

BAL (British anti-lewisite, 2-3 dimercaptopropanol) was shown to counteract the toxic action of metal salts, notably those of arsenic and mercury, a development of great potential importance in chemical warfare injuries, or in the event of the use of phosphorus missiles.

Postwar studies sponsored by the Army Medical Service Graduate School established chloramphenicol in the treatment of scrub typhus, epidemic typhus fever and typhoid fever.

At the end of the period, training in clinical medicine and standards of practice had been much improved by mutual study of problems through the consultant and research systems. Clinical medicine faced the potential problems of the second half of the century much better prepared than at any other time in its history.

NEUROPSYCHIATRY

Neuropsychiatry had not been recognized as a separate war medical specialty prior to World War I. Massive fire power produced much wounding and death in great campaigns, the outcome of which hung in the balance. Battle stress, both physical and mental, was severe. Preventive psychiatry and the range of psychic trauma were not understood. "Shell shock" masked much injury of psychiatric importance.

The development of psychiatric practice in the short war period was rapid. Although all emphasis was upon therapy, one important principle of war evolved: expeditious treatment as far forward as possible. Encouragement, rest, persuasion and suggestion in the combat organization accomplished more than hospital therapy in rear areas.

In World War II neuropsychiatric disorders were the greatest single cause of disability. Approximately 1,000,000 patients were hospitalized, an

admission rate of 45 per 1000 troops per year. This emphatically disproved the conviction indulged by some that psychiatric screening at induction would preclude important psychiatric illness in combat forces. It was found that resistance to stress is relative and varies by individual, and that all are susceptible when the intensity and duration of stress are great enough. Preventive psychiatry developed slowly, and the principle of psychiatric triage and treatment as far forward as possible had to be re-established. In the early period overseas, hospital treatment returned only 5 to 10 per cent of cases to duty; later, division psychiatrists and forward treatment centers salvaged 60 per cent of casualties for combat or service duty within two to five days. Of cases evacuated to rearward hospitals, an additional 30 per cent were saved for noncombatant duty. The principles involved are fundamental to the development of military psychiatry.

Two treatment methods, psychotherapy under narcosis and group therapy, were widely used during the war. Convalescent facilities and neuropsychiatric hospitals provided the opportunity for the critical study of large numbers of cases and the development of treatment technic, the full significance of which cannot yet be evaluated.

One of the great dividends of the war experience was that the military authorities and the medical profession in general came to appreciate the implications of neuropsychiatric disease in modern war. The magnitude of the problem is enormous. It was met with fortitude and great effort by the psychiatric profession. Much remains to be learned, especially in the prevention of psychiatric casualties.

Reconditioning and Rehabilitation

Reconditioning is the improvement of subpar inductees or the temporarily disabled through graduated training to meet the rigorous demands of military duty. Rehabilitation is the fitting of the soldier no longer capable of military duty for social and economic usefulness by maximum hospital treatment, vocational guidance, prevocational training and resocialization. The former conserves manpower; the latter is an obligation of American medicine.

Reconditioning was little practiced in World War I. In World War II a vast program in hospitals, convalescent centers and special training camps returned many thousands to effective service. It employed and expanded physical medicine and utilized the physical trainer, athletic director and psychologist. In World War I chronic disability in the traumatized, tuberculous and mentally ill soldier brought to the fore the necessity for developing an extensive program of physical reconstruction and vocational education. Reconstruction hospitals and special departments in general, base and other types of hospitals showed much development in physical medicine and in the vocational training of the disabled soldier. General education and preparation for the trades for the crippled, the blind, the deaf, the speech defective and the psychiatric casualty characterized the program. The great need for physiotherapy and occupational aides, recreational experts and social-service workers established these ancillary medical activities and provided the training for many who played important roles in medicine in the postwar years.

President Franklin D. Roosevelt directed maximum rehabilitation for the disabled, and charged military authorities with that responsibility in 1944. The program already in being was expanded and eventually included special hospitals for the blind, the deaf and psychiatric casualties. Orthopedic and plastic surgery restored many, and ingenious and cosmetically acceptable prosthetic appliances for amputees and others were major contributions. Paraplegics, formerly accepted as potentially moribund, now have a much longer life expectancy and in some cases have been restored to self-maintenance and even economic independence. The importance of this concept, as developed by Rusk and his associates during and since the war and as an integral part of public health and industrial medicine, has established a new specialty in medicine that gives promise of economic usefulness and independence to many thousands who otherwise would have been helpless wards of society.

Research and Development

Research is fundamental to the preparation of military medicine for war and for the solution of special problems arising during war. Sternberg, in recognition of this, established the Army Medical School and the Tropical Medical Research Board and fostered the investigations of Reed and others. The concept was furthered in World War I by the organization of the National Research Council in 1916, the expanded activities of the Army Medical School and the research programs sponsored by the Research Society of the American Red Cross in Europe.

The global dimensions of World War II, the probability of extensive wounding from vastly improved weapons, climatic stress in the Arctic and tropics and tropical diseases required medical preparation not before contemplated. To meet these problems required the exploitation of all medical resources available to the Surgeons General. There rapidly developed an interservice co-operation that additionally mobilized the scientific facilities of other governmental and civilian agencies that could contribute to the development of means and methods for the health protection and medical care of the American soldier, airman, sailor and marine. A program of scientific research was formulated by committees of the National Research Council, composed of foremost experts in many

fields of medicine and the allied sciences, and was supported by the Committee on Medical Research of the Office of Scientific Research and Development. Research on many problems was promptly initiated in laboratories, universities and government institutions and by individual workers throughout the country. Coincidentally the Army, Navy and Air Force Medical Departments organized research boards which, jointly with the Professional Service Divisions, studied military medical research needs. The early forging of a comprehensive research program gave prompt answers to many urgent problems and thoroughly demonstrated the advantage of mobilizing many scientific disciplines in a co-ordinated research effort.

War research activities brought rich dividends to the military medical services, among the greatest of which was the full recognition that research and development must be an integral part of the military organization. The research and development organizations within the services during and since the war have had much to do with the establishing of better interservice relations and a feeling of mutual responsibility with many segments of civilian medicine.

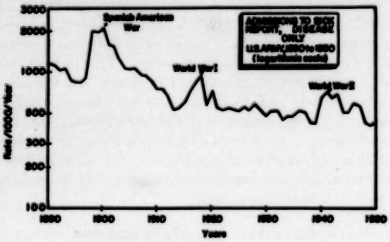

FIGURE 1.

AVIATION MEDICINE

Aviation medicine was recognized as a medical specialty in 1917, when a medical officer was appointed as chief surgeon to the Air Service. In January of 1918 a research laboratory for aviation medicine was activated. Later, the Air Service Medical Research Board became the School of Aviation Medicine at Randolph Field, Texas, and since World War II has been expanded as the Air University. In 1937 a physiologic research laboratory was opened at Wright Field, Dayton, Ohio, where Grow, Armstrong and others contributed much to the development of aviation medicine, especially as related to flying stresses, oxygen requirements, safety devices, physiologic and psychologic responses of man to flying and the adaptation of the airplane to man. These studies, together with the establishment of physical and mental criteria for the selection of pilots and fitness-testing devices, made possible the wide range of successful uses to which the airplane was put in World War II and have materially aided in the development of civil aviation and industrial medicine.

Since the war the Surgeon General of the Air Force has concentrated great research and aeromedical talent on adaptation of man to the high speeds and altitudes of the modern airplane and improved safety in flying; in conjunction with the other medical services he has sought to improve means of health protection and of prevention and treatment of disease and injury.

SUMMARY AND DISCUSSION

In fifty years, military medicine has progressed from an auxiliary and largely unsupported part of the military service to a technically and militarily competent service and staff component of the Armed Forces. The World Wars tested it severely.

In accord with our democratic traditions, a small nucleus of Regular Army medical specialists has combined with the civil medical profession to forge vast and effective organizations in time of war. In peace, an increased sharing of responsibility has eliminated barriers between military and civilian medicine and has fostered the fundamental concept that government agencies represent the will of the people. Proof of the validity of these principles lies in the steady reduction in disease morbidity and fatality in the injured (Fig. 1 and 2 and Table 1).

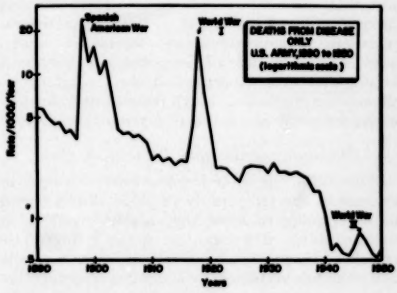

FIGURE 2.

At the end of the first half of the twentieth century, military and civilian physicians alike face great potential problems. Two great world conflicts have wrought many drastic changes in the social fabric of whole nations and of the world.

Destruction was unprecedented, yet these conflicts saw profound and rapid advances in medical science and practice. Military medicine as the practice of mass prevention and treatment of disease and injury has touched the lives of all of us in the past half-century and may do so again as we face the social and economic changes of the second. It is only by the integration of all phases of medical effort that we can face future responsibilities confidently. Mutual understanding, co-operation and co-ordination of effort are the fundamental concepts.

We are indebted to the following for counsel and suggestions in evaluating fifty years of military medical developments: members of the staff of the Surgeon General, U. S. Army, Colonel William S. Stone, M.C., U. S. Army, Brigadier General Oscar P. Snyder, D.C., U. S. Army, The Commandant and staff of the Medical Field Service School, Major General Norman T. Kirk (Ret.), Major General Morrison C. Stayer (Ret.), Major General George F. Lull (Ret.), Brigadier General James S. Simmons (Ret.), Drs. Haven Emerson, Edward D. Churchill, William S. Middleton, William C. Menninger, I. S. Ravdin, Hugh J. Morgan, Stanhope Bayne-Jones and Ralph Muckenfuss.

Bibliography

Absentees in Military Medicine, Science in World War II, OSRD. First edition. 2 vol. 900 pp. Boston: Little, Brown and Company, 1948.

Armstrong, H. G. *Principles and Practice of Aviation Medicine.* Second edition. 514 pp. Baltimore, Maryland: Williams and Wilkins Co., 1943.

Ashburn, P. M. *A History of the Medical Department of the United States Army.* 448 pp. Boston and New York: Houghton Mifflin Co., 1929.

Beecher, H. K. *Resuscitation and Anesthesia for Wounded Men: The management of traumatic shock.* 161 pp. Springfield, Illinois: Charles C Thomas, 1949.

Churchill, E. D. American surgeon, A.U.S. *Surg., Gynec. & Obst.* **84:** 529-539, 1947.

Idem. Surgical management of wounded in Mediterranean theater at time of fall of Rome. *Ann. Surg.* **120:**268-283, 1944.

Idem. Trends and practices in thoracic surgery in Mediterranean theater. *J. Thoracic Surg.* **13:**307-315, 1944.

Developments in military medicine during administration of Surgeon General Norman T. Kirk. *Bull. U. S. Army M. Dept.* **7:**520-562, and 594-646, 1947.

Garrison, F. H. *Introduction to the History of Medicine.* Fourth edition. 996 pp. Philadelphia and London: W. B. Saunders Company, 1929.

Idem. Notes on history of military medicine. *Mil. Surgeon*, 1921-22.

Hanson, F. R. Combat psychiatry. *Bull. U. S. Army M. Dept.* **9** (Supplemental Number): November, 1949.

Havard, V. *Manual of Military Hygiene for the Military Services of the United States.* 481 pp. New York: William Wood and Co., 1909.

Inter-allied Conferences on War Medicine, 1942-1945. Edited by H. Tidy and J. M. B. Kutschbach. 531 pp. New York: Staples Press, Ltd., 1947.

Long, A. P., and Sartwell, P. E. Tetanus in United States Army in World War II. *Bull. U. S. Army M. Dept.* **7:**371-385, 1947.

Menninger, W. C. Psychiatric experience in war, 1941-1946. *Am. J. Psychiat.* **103:**577-586, 1947.

Middleton, W. S. Medicine in European Theater of Operations. *Ann. Int. Med.* **26:**191-200, 1947.

Military Medical Manual. Fifth edition. Harrisburg, Pennsylvania: The Military Service Publishing Company, 1942.

Simmons, J. S. Present state of Army's health. *J. A. M. A.* **122:**916-923, 1943.

Idem. Preventive medicine program of United States Army. *Am. J. Pub. Health* **33:**931-940, 1943.

Idem. U. S. Army Medical Department Research Board. *Mil. Surgeon* **67:**479-489, 1930.

Idem. United States Army's war in air against mosquito-borne diseases. *Am. J. M. Sc.* **196:**153-167, 1938.

War Department: The Medical Department of the United States Army in the World War. Washington, D. C.: Government Printing Office, 1923, 1926, 1928, 1929, 1927, 1927. Vol. I, VI, IX, X, XI, Part I, XIII, Part I.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 37161

Presentation of Case

A fifty-year-old man entered the hospital because of severe shortness of breath.

One month before admission, the patient, who had been well previously except for minimal tiredness, developed aching in the arms and legs, generalized weakness, extreme fatigue and symptoms of a mild "cold." At this time he also experienced some pain in the chest both to the right and to the left of the sternum that was dull in character and was not related to respiration. Three weeks prior to admission the chest pain became severe and pleuritic in character, and he developed rather pronounced shortness of breath. A "rub" was found on examination by a physician, and the patient was admitted to another hospital. Electrocardiograms were said to have shown low voltage in all leads and inverted T waves. A chest film allegedly showed "scar tissue" at the root of the lungs, which raised the question of tuberculosis. Gastric aspirations were negative. The sedimentation rate was said to have been elevated. Over a period of two weeks the patient gradually improved on bed rest and aureomycin therapy and was discharged eleven days prior to admission to this hospital. Four days prior to admission another electrocardiogram was said to have shown improvement over previous tracings. The physician could no longer hear the rub and stated that his "blood test" was improved. That evening, however, dyspnea returned. It was mild at first and present only on exertion but became progressively worse. Two days before entry it was present even when he was at rest in bed. The other symptoms he had noted were some tense swelling of the abdomen, aches in the shoulders on motion and questionable ankle edema one day before admission. In the eighteen hours prior to admission he received 1.2 mg. of digitoxin.

He had had osteomyelitis of the right elbow at the age of seventeen. A duodenal ulcer recognized eleven years ago was treated medically and was checked repeatedly in the interim. There was no family history of tuberculosis.

On physical examination the neck veins were engorged and pulsated almost to the angle of the jaws. The chest showed prolongation of the expiratory phase of respiration. There were signs of a massive right pleural effusion with a flat percussion note, decreased tactile fremitus, vocal fremitus and breath sounds over the right lung field. There was questionable fluid at the left base as well. Atelectatic rales

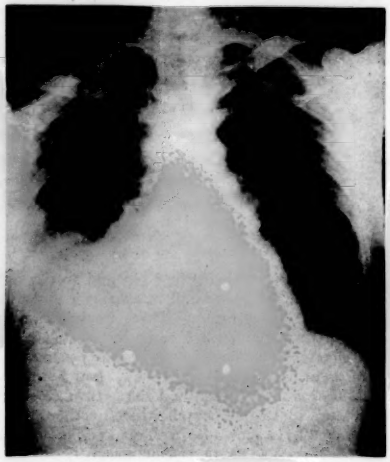

FIGURE 1.

were heard over the fluid on both sides. The heart was enlarged 2 cm. to the left of the midclavicular line in the fifth and sixth intercostal spaces. There was normal sinus rhythm, at a rate of 138 per minute with decrease in amplitude of sounds. The aortic second sound equaled the pulmonic. Marked pulsus paradoxus was present. Superficial veins over the chest, especially on the right side, were dilated. There was some congestion of the skin over the chest. The abdomen was tense and rounded and there was slight pouting of the umbilicus. The liver was felt 5 cm. below the costal margin in the midclavicular line (consistence and tenderness not mentioned). There was no shifting dullness or fluid wave. There was an old scar and deformity of the right elbow.

The temperature was 100°F., the pulse 120 and the respirations 30. The blood pressure was 115 systolic, 70 diastolic.

Examination of the blood revealed a hemoglobin of 12.6 gm. and a white-cell count of 11,700, with 86 per cent neutrophils. The blood Hinton test was negative. The sodium was 134.9 milliequiv., the chloride 93 milliequiv. and the carbon dioxide 25 milliequiv. per liter; the nonprotein nitrogen was 27 mg. per 100 cc. and the sedimentation rate 10 mm. in fifteen minutes, 28 mm. in thirty minutes, 38 mm. in forty-five minutes and 46 mm. in sixty minutes.

X-ray examination of the chest showed a coarse, strand-like increase in density in both lung fields. Fluid was present in both pleural cavities, more marked on the right side (Fig. 1). There was a calcified nodule on the apical segment of the right upper lobe and also a patchy area of increased density in this segment. The heart was enlarged and somewhat globular in shape. The cardiophrenic angle was obscured on both sides. A plain film of the abdomen showed slight liver enlargement. An electrocardiogram revealed a normal rhythm at a rate of 110 with PR intervals of 0.14 second. The T waves were inverted in Leads 1, 2, V_4, V_5 and V_6, flat in Lead 3 and upright in Leads V_1 and V_2. QRS complexes were normal.

On the day of admission 1100 cc. of slightly yellow, cloudy fluid was removed from the right chest. The cytological report on this specimen was doubtful. The fluid was also sent to the laboratory for culture and guinea-pig inoculation. The gastric washings were reported negative for acid-fast bacilli on smear. On the second hospital day the patient developed an apical gallop rhythm uncontrolled by digitoxin (0.1 mg. per day). Later in the day a friction rub was heard over the right chest anteriorly, with absent breath sounds over the lower part of the right chest. The temperature ranged between 100 and 101°F. Additional information was obtained at this time that the patient apparently had had increasing fatigue for at least six to eight months prior to admission, that for three or four months he had had difficulty in keeping up with the younger men at work due to shortness of breath and that approximately two months before admission he had developed a slight cough. Fluoroscopic examination showed decreased amplitude of cardiac pulsations. On the third hospital day the patient was started on streptomycin and penicillin therapy. The following day he had become quite anorexic, so that he was able to eat only soft food; he was very pale. The next day the pulse fell to 60 for a few hours but returned to 120. An operation was performed on the sixth day.

DIFFERENTIAL DIAGNOSIS

DR. RICHARD J. CLARK*: Let us review the highlights of this history. In the first place, the pa-

*Assistant physician, Massachusetts General Hospital.

tient on further questioning admitted that he had not been feeling well for a period of six to eight months. He had had gradually increasing difficulty, with signs that pointed toward either heart or lungs, with an associated dyspnea. A month before admission he had some exacerbation of these symptoms and an episode described as generalized weakness, extreme fatigue and symptoms of a mild cold. The possibility of one of the prevalent viral infections with pulmonary involvement is suggested. After this he experienced chest pain that was somewhat vague at first and after a week obviously pleuritic. A rub was found, but I am not told whether it was pleural or pericardial in nature. An electrocardiogram was reported consistent with pericarditis. It may be fair to assume that the rub was pericardial in origin.

The findings in the x-ray films made in another hospital apparently raised the question of tuberculosis. Gastric aspirations were done, but with negative results for acid-fast bacilli. Aureomycin therapy was started. I may speculate that at this point the diagnosis rested between tuberculous and viral pericarditis and that as there was no proof of the former a trial of therapy for the latter was instituted. It is of interest that nothing was said about pleural fluid's being seen in the x-ray films at that time. He appeared to improve after ten days of therapy but for only a short while, as a week later he developed a rapid progression of symptoms with very severe dyspnea, evidence of abdominal congestion and probable ankle edema. He was digitalized, but apparently received very little benefit from this; such is not surprising with mechanical obstruction in the presence of pericarditis.

On entry to this hospital physical examination showed signs that were entirely consistent with a pericardial effusion and cardiac tamponade. There was a fairly large amount of fluid in the right chest and some in the left. There was enlargement of the liver and possibly abdominal fluid. The laboratory findings were consistent with low-grade infection, the white-cell count being up to 11,700 with 86 per cent neutrophils and the sedimentation rate being definitely elevated. The electrocardiogram as described was entirely consistent with pericarditis.

May we see the x-ray films at this point?

Dr. Stanley M. Wyman: The original films of the chest show a rather large quantity of fluid in the right pleural space and a smaller quantity in the left (Fig. 1). Both lower lobes are somewhat atelectatic, probably due to compression by the fluid. The heart shadow appears larger than normal but smooth in contour. Scarring is present in the apex of the right lung and there is a definite focus of calcification there. In addition, there is some indefinite fullness in the apex of the right lung or in the mediastinum adjacent to the lung. This was believed by some observers to indicate enlarged lymph nodes in this region. The hilar shadows are definitely enlarged, and there is a coarse strand-like increase in density radiating out from both hili into the lung fields, with some fine linear streaking in the extreme peripheral portion of the lung. This coarse, strand-like density suggests vascular shadows, but whether they are arterial or lymphatic it is difficult or impossible to say. The liver appears to be enlarged, but it is not possible to outline its upper margin; the lower margin is also very poorly seen. No other definite abnormality is recognized in the abdomen. Two later films of the chest, taken five and seven days after the original examination, show progression of the process in the chest and a more definite suggestion of mediastinal lymph nodes, or possibly a mass in the medial portion of the right lung. Fluid in the left chest has increased considerably.

Dr. Clark: Has the size of the heart increased?

Dr. Wyman: It is difficult to be sure, because the original films were taken upright at a six-foot distance, so that there is considerable error from magnification.

Dr. Clark: Does this increase in density look more like glandular or lung-parenchymal involvement?

Dr. Wyman: I cannot be sure. Some observers thought it was lymph-node involvement; one observer thought it was the lung itself. I tended to think it was probably lymph nodes.

Dr. Clark: The appearance of the x-ray films themselves gives me a different impression from the written description. I had anticipated seeing a considerably larger cardiac silhouette than is evident in these pictures.

After these examinations a chest tap was done on the right side, with removal of 1100 cc. of slightly yellow, cloudy fluid. I am told nothing about the cell count and specific gravity. Cytology reports were doubtful. The fluid was cultured, but I do not know what the results of the cultures or guinea-pig inoculations were. The patient developed a gallop rhythm at the apex with a fairly rapid rate that was not controlled by digitoxin. Along with this he had increasing shortness of breath, he was unable to swallow or to eat well and he appeared to be failing rather rapidly. On the sixth day an operation was performed. What was the operation? I shall assume that this was for the purpose of pericardial biopsy and drainage.

Our chief problem is to determine the cause of the cardiac tamponade along with that of the chest fluid. Almost any process that involves the pericardium may also involve the pleura and vice versa, be it infection or tumor. With which are we dealing here? The possible mass developing in the right medial lung, as described in the x-ray report, could well be consistent with tumor, but the description was not definite. The marked tamponade

without a greater degree of pericardial fluid than was apparent could argue for malignant involvement. The absence of blood in the pleural fluid is somewhat against tumor. The cytologic report is not of any positive help. If this process was tumor, what was its type? Mediastinal lymphoma is a possibility. I must certainly consider metastatic carcinoma, but there is no clue in the history or examination to its possible origin. Primary carcinoma of the lung may spread to the mediastinal lymph nodes and pericardium.

If I am dealing with infection rather than tumor, what type is most likely? Rheumatic fever with pericarditis is to be eliminated on the basis of absent valvular disease and other rheumatic stigmas. I mentioned viral or idiopathic pleurisy in discussing the early stage of the disease. Viral pericarditis may occur in association with a virus pneumonia, but it does not usually produce so severe a cardiac tamponade or so prolonged a course as is shown here. What about pyogenic infection involving the pleura and pericardium? With such a process I should have expected a more fulminating course and a more violently ill patient. Had a pyogenic infection been demonstrated by smear or culture, immediate surgical drainage would have been indicated — and this was not done. We are now coming to realize that tuberculous pericarditis is perhaps more common than generally thought, and it must be seriously considered in the instance of any such process that does not clear fairly promptly. It may be associated with pulmonary, pleural or miliary tuberculosis. This patient had an old calcified gland, and changes in the x-ray films that could have been consistent with tuberculous infection. Tuberculosis of the pericardium is a considerable bugaboo, as it is a diagnosis that is almost impossible to make clinically. Today, with the availability of streptomycin, a positive diagnosis becomes more important than in the past. For this reason, and because of its relative simplicity and safety, pericardial biopsy is now advocated in such cases as the one means of making a definite diagnosis. In the past, results of guinea-pig inoculation have been relatively disappointing. In this case I believe that this was the logic followed in arriving at a clinical diagnosis of tuberculosis, which will be my first choice. As a second choice, malignant disease involving the mediastinum, the pleura and the pericardium is to be seriously entertained. There was some evidence of myocardial involvement and possible conduction defect, but such is more the rule than not in the presence of severe pericardial disease. The abdominal findings were in all probability due to passive congestion secondary to the cardiac tamponade.

Dr. Alfred Kranes: How do you explain this appearance higher up on the basis of tuberculosis? In the last two films it looks like compression. Do you think it is a tuberculous infiltration?

Dr. Clark: I wondered about that. The radiologists are not too certain about it. That is an argument in favor of tumor rather than tuberculosis.

Dr. Kranes: I wonder if he did not have a carcinoma of the bronchus with pericardial involvement from the pleura.

Dr. Clark: That is a possibility.

Clinical Diagnosis

Tuberculous pericarditis.

Dr. Clark's Diagnosis

Tuberculous pericarditis.

?Malignant disease of mediastinum, pleura and pericardium.

Anatomical Diagnosis

Adenocarcinoma of pancreas with metastases to pericardium (with cardiac tamponade), lungs, mediastinal lymph nodes and adrenal glands.

Pathological Discussion

Dr. Lamar Soutter: We operated on this patient with the same diagnosis that Dr. Clark made. Our first choice was tuberculosis; our second was tumor. The preliminary report on the films, made when they were wet, did not suggest the presence of tumor. This patient was deteriorating quite rapidly. We decided to do a therapeutic pericardial tap to relieve his tamponade. We were prepared to make a pleural pericardial window in order to produce a complete decompression of the heart if it were necessary. We introduced the needle below the xiphoid, went through what we thought was pericardium and withdrew whole fresh blood that was somewhat dark and not under much pressure. The fact that blood was obtained suggested that we had gone into the ventricle; the fact that it was not under pressure indicated that we had not. We tapped the pericardium anteriorly in two other places and obtained the same dark blood. I then talked to his physician and his cardiologist, because I thought the patient's condition was so bad that he would not live if I merely returned him to the ward after removing a few cubic centimeters of blood, and I believed that I should make an adequate opening so that the pericardium could drain until we could control the source of bleeding, presumably carcinoma, by radiation therapy. I believed that the blood came from metastatic disease in the pericardium and that the primary lesion probably was in the lung. We made a small incision in the chest cavity at the fifth left intercostal space and exposed dark, thickened pericardium. I put a needle into the pericardium to a depth of about an inch before I struck the bloody material that I had aspirated previously. In order to be certain of the nature of the pericardial disease I took a specimen for biopsy. A frozen section

showed carcinomatous infiltration. I made a window somewhat gingerly, because I was not sure where the pericardium stopped and the myocardium began; however, I managed to enter the pericardium, which contained a good deal of bloody material that was not under much pressure. In some areas the pericardium and myocardium were densely adherent. I was afraid to break up the adhesions because of the invasion of the heart wall. We did not explore the chest any further. We sucked out the fluid, put a catheter in the chest so that the

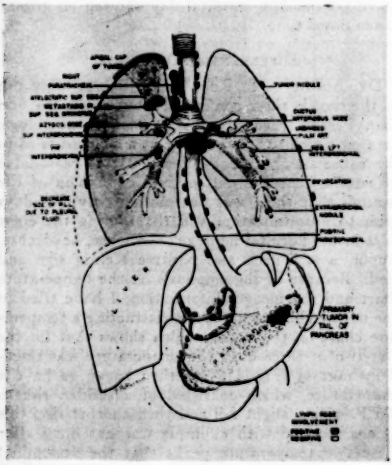

FIGURE 2.

fluid could drain and sent him back to the ward. He was in very poor condition the next day. Because the tamponade was unrelieved we believed that there was probably considerable constriction of the pericardium around the heart where it was adherent. We also thought that enlarged lymph nodes in the region of the superior vena cava had caused it to be constricted. He stayed alive for two or three days, but he was at no time well enough so that we could give him x-ray therapy. Usually patients with pericardial effusion due to tumor are relieved enough by a pleuropericardial window that they can be given x-ray therapy, which checks the effusion. Two such patients treated here have become well enough to go home; one is back at work.

DR. BENJAMIN CASTLEMAN: The Radiology Department has become much interested in the observation of lymph nodes in the chest. They had this drawing (Fig. 2) made of what was found at autopsy. The large lymph nodes probably made up the shadow that is in the medial portion of the right upper chest. There was also a cap of tumor over the apical segment, which was collapsed because of bronchial obstruction by enlarged lymph nodes. Microscopical examination of this bronchus showed tumor infiltrating a portion of mucosa, but it seemed to be not a primary tumor but one that came from without, although that was hard to be certain of. A similar nodule of tumor surrounding a bronchus was found in the left lower lobe and there were numerous tumor nodules over the pleura, with pleural fluid on both sides. As you can see from the diagram, there were nodules up and down the aorta in the region of the stomach and in the region of the pancreas. Most of the lymph nodes were replaced by tumor. In the tail of the pancreas we found a good-sized nodule, which I believe was the primary tumor. It is well known that carcinoma of the pancreas often metastasizes to the mediastinal lymph nodes. In fact, in a few of our cases the mediastinal metastases had been called Hodgkin's disease before the autopsy showed that the primary lesion was in the pancreas. The pericardium and heart together were very massive, weighing about 1000 gm., more than half the weight being due to carcinomatous infiltration of the pericardium. Carcinoma involved not only the parietal pericardium but also the surface of the heart, the visceral pericardium measuring about 3 cm. in thickness. It extended into the myocardium in places and may account, as Dr. Clark mentioned, for the lack of good conduction. There were a few metastases to the adrenals but none to the liver, which showed a central congestion perhaps due to the cardiac tamponade. Microscopically, the lesion in the pancreas was a classic adenocarcinoma. The metastases were less well differentiated; in the lung they showed no acinar arrangement.

CASE 37162

PRESENTATION OF CASE

A fifty-seven-year-old man was admitted to the hospital because of constipation and abdominal cramps.

The patient was well until one year before entry, when he began to pass small amounts of blood by rectum. Three months before admission he noted a decrease in the caliber of the stools. Five days before admission he was unable to have a bowel movement and shortly afterwards developed moderately severe generalized abdominal cramps. The patient had one small movement two days before entry, with some blood, and on the night before admission passed a large amount of gas, with considerable relief of the cramps.

The past history was noncontributory.

On physical examination the heart and lungs were normal except for a slight apical murmur. The abdomen was slightly distended, but there were no masses or tenderness. Occasional loud peristaltic rushes could be heard. On rectal examination an annular, nodular and tender lesion was palpated about 8 cm. within the rectal canal.

The temperature, pulse and respirations were normal. The blood pressure was 140 systolic, 70 diastolic.

The urine was normal. Examination of the blood showed a hemoglobin of 12.5 gm. per 100 cc. and a white-cell count of 10,200. In the hospital the patient complained of increasing abdominal cramps. The abdomen remained distended but was not tense or tender, and peristalsis was normal. There were no bowel movements during the first three hospital days but the patient did pass small amounts of gas from time to time. A proctoscopic examination disclosed a fungating, annular lesion that bled easily and completely encircled the bowel wall. An intravenous pyelogram was unsatisfactory. The biopsy of the rectal lesion was reported as adenocarcinoma, grade II. A Miller–Abbott tube was passed.

During the first two hospital days the temperature curve showed single spikes to 100°F. On the following day it rose to 101°F. and on the next day suddenly rose to 105°F. Associated with this was a pulse rate of 120 per minute and a respiratory rate as high as 30. Several hours later the temperature had dropped to 102°F. Still later in the day the patient complained of severe abdominal cramps, shortly after which he had a shaking chill. The temperature again rose to 105°. During these episodes repeated examinations failed to reveal evidence of increased abdominal tenderness or spasticity. Peristalsis remained normal. Another rectal examination did not disclose any increased tenderness about the tumor mass. It was felt that decompression was urgently needed, and later the same day a loop transverse colostomy was done under spinal anesthesia. A white-cell count was recorded as 15,100. On the fifth hospital day the temperature was 104°F. and the pulse 100. The patient complained of severe burning pain in the low back and buttocks. There was no costovertebral-angle tenderness and the abdominal findings were unchanged. The loop colostomy discharged copious amounts of gas and feces. There was no chest pain. On the sixth hospital day the temperature was 102°F., and the patient became irrational, requiring restraints. A roentgenogram of the chest was negative. A plain film of the abdomen showed large amounts of gas in both large and small bowel. Twenty-four hours later the temperature had dropped to normal, with a single spike late in the day to 100°F. The patient complained of headache but was more rational and co-operative. A neurologist found stiffness of the neck, a positive Kernig sign and a drooping right lid. A spinal tap revealed bright orange-yellow spinal fluid under a pressure of 130 mm. of water. There were 970 red blood cells and 267 white blood cells per cu. mm. The protein was 2250 mg. per 100 cc. and the sugar 37 mg. per 100 cc. The following day the physical findings were generally the same, but the patient was moderately stuporous. The white-cell count was 10,400. The temperature rose to 107°F., and the patient died.

Treatment in the hospital consisted of one blood transfusion, penicillin and streptomycin during the fifth and sixth hospital days and crysticillin during the last three days of life. A blood culture taken on the fifth hospital day showed no growth in ten days' time.

Differential Diagnosis

Dr. Augustus S. Rose*: One is inevitably put on the spot in these conferences, but this is the first time I have been given such meager neurologic data on which to try to arrive at a conclusion as to the cause of a patient's death. It is reasonable to assume that the patient had carcinoma of the rectum, that there was obstruction only five days prior to hospitalization and that during the eight days in the hospital he developed fever, headaches, stupor, a stiff neck and positive Kernig sign and died. Because of the emphasis on the temperature chart and the meager information, I have tried to line up the chronology by reconstructing a temperature chart on the board, which shows that for the first two or three days the temperature was essentially normal (Fig. 1). He then began to have a temperature, which consisted of a sudden rise to 105°F. with a slight fall and then another rise, this time associated with chill. It was not until after these two temperature peaks that the abdominal operation was performed and not until after that, as indicated by the repeated comments in the record, that it was felt reasonably sure that the patient did not have peritonitis. At the time of the operation he apparently was given a transfusion and was put on penicillin, and then his temperature fell. Concerning whether there is any correlation other than the chronology, I cannot, of course, say. In addition, he was given spinal anesthesia, after which he had pain in the back and buttock. On the next day he developed delirium requiring restraint, and on the following day he had headache, stiff neck and a positive Kernig sign. He also was found to have ptosis. A lumbar puncture was done; the initial pressure was low — 130 mm. He then had stupor and a terminal precipitous rise in temperature and died.

I believe the sudden rise in temperature prior to the operation may have been a complication induced in the hospital by the procedures that had been done, namely, the routine examinations, proctoscopy, x-ray examinations, including

*Associate neurologist, Massachusetts General Hospital.

the intravenous pyelogram, which was unsatisfactory, and the biopsy of the tumor. The question therefore immediately arises whether the patient developed, either from the intravenous pyelogram or from the biopsy, a thrombophlebitis with subsequent septicemia, which was at least partly controlled by the chemotherapy. I do not believe that I can consider that the abdominal operation per se led to the patient's death in any way, except for the possibility that spinal anesthesia in this particular patient may have had some reasonable importance. Considering that the spinal fluid was red-yellow with a total protein of 2000 mg. three days after the spinal anesthesia, I wonder how the spinal fluid could have been clear and colorless at the time of administering the spinal anesthesia.

With a total protein showing that much elevation and that color, one must assume that the patient had a block, even though there was an initial pressure of only 130 mm. of water. There was no information about the reflexes and no sensory examination to help me make a reasonable guess, but with the known carcinoma and with a total protein of 2000 mg. even though he had a spinal anesthesia, I think I must assume that this patient had metastatic disease involving the spinal canal and that there was a chemical block even if there was no dynamic block. The chemical block or even the dynamic block of the spinal canal could not cause his death within so short a time. I must also take into consideration that the patient had developed delirium one day or a day and a half after the spinal anesthesia and operation, the subsequent day developed a headache and a stiff neck and drooping of at least one lid, and the next day became stuporous and died. This suggests without any further data, in fact points vividly to the fact, that this patient had intracranial disease and that his death was cerebral.

The question arises of the nature of the cerebral lesion or lesions. The temperature chart leads me to raise first the matter of infection and secondly the question whether he had metastatic disease. On the basis of the data at present I cannot distinguish between these two. On the other hand, the spinal-fluid cell count showed 200-odd white cells but also some red cells. The sugar was 37 mg. Certainly the degree of infection below the block was not very great. If it was a bacterial infection, the sugar should have been much lower and there should have been a much higher white-cell count in the spinal fluid. I do not believe, therefore, that I can assume that the spinal anesthesia carried an infection into the spinal canal. Is it reasonable at this point, Dr. Mallory, to ask whether or not reflexes were examined and whether or not there was a sensory level?

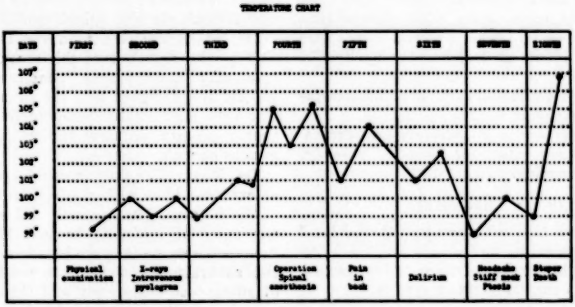

FIGURE 1.

DR. TRACY B. MALLORY: Dr. Timberlake, can you give us the details?

DR. W. H. TIMBERLAKE: The sensory examination was negative.

DR. ROSE: What about the reflexes and pupils?

DR. TIMBERLAKE: They were all normal. The ptosis was minimal.

DR. ROSE: Perhaps I should not have asked. The white-cell count before operation was 10,000; it was 15,000 on the day after the operation, but it subsequently fell. I would have to say that I believe that this patient had carcinomatosis, that is to say, metastatic disease involving the cerebrospinal axis, and that he probably died of increased intracranial pressure, which was not shown below because there was a spinal block. That puts me out on a limb that I cannot support with the data, but I cannot make a diagnosis otherwise. If he had a thrombophlebitis involving one or more of the venous sinuses or if he had a meningitis about

this hypothetical block, I have no way of making such a diagnosis.

DR. MALLORY: Are there any questions or comments?

DR. ALFRED KRANES: If the onset was associated with an actual chill, it seems to me one would be forced to make a diagnosis of some sort of infection.

DR. MALLORY: I think it was believed to be a chill.

DR. KRANES: Do you not think that if the patient actually had a real shaking chill that is just as important as the spinal-fluid sugar?

DR. ROSE: The temperature chart showed that he actually had an infectious process, but where was it? Was it in the central nervous system? I cannot say so.

A PHYSICIAN: Why should carcinoma or metastasis in the brain result in sudden death?

DR. ROSE: Increase in intracranial pressure associated with some infectious process, possibly, but I am only guessing.

CLINICAL DIAGNOSES

Carcinoma of rectum with perforation and local abscess formation.
?Subarachnoid hemorrhage.
?Metastases to central nervous system.

DR. ROSE'S DIAGNOSIS

Metastatic carcinoma involving central nervous system.

ANATOMICAL DIAGNOSES

Carcinoma of rectum, with metastases to liver.
Perirectal abscess.
Lumbosacral epidural abscess.

PATHOLOGICAL DISCUSSION

DR. MALLORY: The autopsy of the body cavities showed nothing of great importance except the known carcinoma of the bowel, which had extended through the wall into the perirectal tissues and around which there was some degree of abscess formation. There was a single metastasis in the liver. I shall ask Dr. Kubik to tell us about the findings in the central nervous system.

DR. CHARLES S. KUBIK: I agree with Dr. Rose that the clinical data were exceedingly meager — and it may be added that they were not too characteristic of the condition that was found. There were no metastases to the spine or to the spinal epidural space, but there was a collection of pus outside the dura covering the lower part of the cord and the cauda equina. There was no grossly visible meningitis, that is, subarachnoid exudate, of the spinal cord or brain or any visible infection of the vertebras, and it was impossible at autopsy to trace the pathway of infection between the retroperitoneal tissues and epidural space. On microscopical examination there were, in addition to the epidural infection, regions of necrosis and polymorphonuclear infiltration of the dura, and very small amounts of subdural and subarachnoid exudates. The microscopical findings indicate that after reaching the epidural space the infection had extended through the dura and into the subdural and subarachnoid spaces. Putting that and the clinical course together, I should agree with Dr. Rose that the retroperitoneal infection was probably present before the operation and that the infection extended along thrombosed veins to the epidural space. A similar condition was observed here a number of years ago in the case of an infection spreading from the mediastinum to the spinal epidural space.

DR. MALLORY: What do you think of the possibility, Dr. Kubik, that this second lumbar puncture may have merely penetrated the epidural abscess and not gone into the subarachnoid space? The very high total protein might have suggested that possibility, although the cells do not seem numerous enough.

DR. ROSE: The fluid is certainly consistent with an extradural block of an infectious nature.

DR. KUBIK: That actually did happen in one of our cases of epidural abscess. When the spinal puncture was made, pure pus was obtained before the subarachnoid space was reached. Thus the diagnosis was made early and the patient was operated on with a good result. I agree that in this case the cell count was too low.

Were the dynamics tested?

DR. TIMBERLAKE: I think not. I think only the pulse and respiratory oscillations were recorded.

DR. ROSE: Why did the patient die?

DR. KUBIK: I think the question might be answered by Dr. Mallory. This patient was quite ill. He had an obstruction of the bowel; that alone can account for a great deal. Added to that was infection. Would that not be enough?

DR. MALLORY: I do not think we have a very adequate explanation of why the patient died exactly when he did. He was a very sick man, with metastatic carcinoma and sepsis, and this was an extradural abscess of considerable extent.

DR. ROSE: Are sensory examinations usually normal in patients with an epidural abscess?

DR. KUBIK: Most of the patients have been admitted a considerable time after the onset of the symptoms. In these patients, paresis or paralysis, reflex changes and sensory impairment were usually present and probably would eventually have developed in this case. As a matter of fact, even in this case there were microscopical findings indicative of a severe, acute degenerative process, consisting of swelling of the nerve fibers of the spinal nerves and cord and shrunken anterior horn cells with pyknotic nuclei. The neurologic examination was made twenty-four hours or so before the patient died. It is possible that some abnormal neurologic findings might have developed subsequently. There had been pain in the buttock and leg, which was probably due to involvement of spinal nerves.

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NOT ONLY UNTO CAESAR

Two letters, each written by a distinguished and respected fellow of the Massachusetts Medical Society, appear elsewhere in this issue of the *Journal*. One is addressed to the administrative officers of the Society; the other is in answer to it.

The first letter is from Dr. Charles A. Janeway, professor of pediatrics at Harvard Medical School, who, protesting against the policies of the American Medical Association and the dues that necessity required it last year to levy on its members, has nevertheless paid the dues on the basis of good citizenship. In his letter Dr. Janeway states in passing his reason for payment: the acceptance of the democratic rule of the majority. His real purpose in writing is to give the reasons for his protest. Dr. Earle M. Chapman, a delegate from the Massachusetts Medical Society to the American Medical Association, has written a reply to Dr. Janeway that appears to answer a number of the criticisms of the Association that have been given such wide publicity.

Many members of the American Medical Association, less scrupulous than Dr. Janeway, are still delinquent in paying their 1950 dues to the organization. Some of these members have perhaps failed to pay because they have forgotten the indebtedness, some because they have found it inconvenient or even pecuniarily impossible; others, no doubt, simply are not interested in an investment that seems to bring them no immediate return for their money.

Of special importance to the future position and prestige of organized medicine is the stand taken by those responsible physicians who have not paid as a matter of principle, or, having paid, hold nevertheless to the convictions on which their disapproval of the Association's policies is based. They merit the utmost respect, and their opinions should be studied; they cannot be disregarded if that solidarity so necessary to the continued progress of medicine is to be achieved.

So far as the actual payment of dues is concerned, the only principle at stake is that of personal financial responsibility. This fiscal requirement of the Association is not a matter of an arbitrary assessment for a specific purpose, of which the protestant may or may not approve on "principle," as was the case with the voluntary assessment of 1949, imposed for the purpose of financing the "educational campaign"; it is a matter of paying the legitimate annual dues established by the House of Delegates for the support of an association in which the person concerned holds membership. The policies that seem to require clarification are a thing apart from the reasons for which certain members of such an association may prefer to be dropped for nonpayment of dues than to resign, while in good standing, from the organization that appears to be no longer useful to them.

One of the points on which Dr. Janeway disagrees with the American Medical Association is the presumedly basic tenet that only physicians are com-

petent or entitled to plan the organization of medical care. If this actually were a basic tenet of the Association, then any physician would be justified in rejecting it.

Dr. Janeway's second point is his disapproval of the tactics that the Association "has followed for a number of years of working *against* 'socialized medicine' instead of *for an aggressive program* of its own." Here again, so far as Dr. Janeway's premise is correct, any physician is justified in agreeing with him, and the battle against politically controlled medicine has certainly had many of the aspects of a defensive action.

Dr. Chapman, however, by virtue of his membership in the House of Delegates and because of his familiarity with the policies as well as the diverse activities of the American Medical Association, is in a position to present some reasons for believing that many of the critics of the Association do not see the picture as a whole. Preoccupied with their obsessive condemnation of the blunders and ineptitudes of which the Association may have been guilty, they ignore or discount heavily the great contributions that it has made and is making to the improvement of medical care.

Delighted, apparently, at the opportunity of denouncing a leadership that has seemed in retrospect to have failed so often and so egregiously, they are not yet fully aware of the gradual change that is taking place in that leadership. The American Medical Association still makes mistakes — a not uncommon failing — but it is also still being castigated for the mistakes of a previous era.

It is possible only to repeat the plea that has already been made so often to those who are disappointed or outraged at the real and fancied shortcomings of their national association — to give aid and encouragement to those who are working from within for its betterment.

Before denying it, before branding it publicly, before undermining it by the refusal of financial support, they should ponder the ultimate effect on their profession of such actions. They should make no compromise with what appears to be wrong, but they should study the circumstances carefully to avoid snap judgments and then follow the course that seems most likely to help in the correction of any faults that may be found.

HEADS OR TAILS?

A SCHOLARLY correspondence has come to light in the columns of the *British Medical Journal* regarding the position in which "floaters" are found when taken from the Thames. The original observation (November 18, p. 1172) by Dr. Eric Gardner, a man with wide waterside experience, was that women float face downward; men, face upward.

This argument was sustained by W. Baly Peacock, writing in the issue of December 2 (p. 1277), who refers to both Pliny the Younger and Sir Thomas Browne as added authority in favor of the prone position for females. Hugh Willoughby, however, in the same issue and on his own repeated observation asserts that men only float face down; women, according to the testimony of various watermen, present the obverse.

The mild controversy was carried still further by H. D. Livingstone-Spence (December 16, p. 1389) who had personally observed, from the Waterloo Bridge, the body of an elderly gentleman, clad in a Prince Albert coat, floating face upward. This observer contributes the suggestion that the manner of death might influence in some way the manner of floating: that is, did he fall or was he pushed?

J. R. Salmond, in the same issue, confirms the first-hand testimony of Dr. Willoughby by his own extensive experience, gained beside the ponds of England and of India and along the banks of the muddied Yangtze River. Female bodies float face up and male bodies bottoms up, at least in fresh water. This seems to settle the matter, with the usual exceptions.

The discussion vaguely reminds one of certain idle rumors concerning the attitude that stone knights affect on their sarcophagi — if their toes are up they had been to the Holy Land, if down, they had not trod that sacred soil — hence the well known paraphrase, after Coleridge:

> The knight's bones are dust,
> And his good sword rust;
> His soles they are turned up, we trust.

LITTLE LIVER PILLS

THE Federal Trade Commission, according to an Associated Press dispatch of April 3, has finally ordered the manufacturers of "Carter's Little Liver Pills" to omit the word "liver" from the name and possibly from the advertising copy of these allegedly

diminutive restoratives of health for which such fantastic effectiveness is claimed.

Carter's Little Liver Pills, in fact, as the emphatically expressed conclusion of the decision states, are "nothing more than an irritative laxative compound," and their producers are hereinafter enjoined from stating without qualification that its pills are what one needs when one is "down-and-out, blue, listless, fagged out, down-in-the dumps, irritable, bilious or sullen."

The decision actually does not state unequivocally whether the objection to "liver" is in relation to the qualifying adjective "little" or to the pills themselves. Any assumption, however, that the pills, small or large, are effective only in the treatment of little livers is unwarranted, nor is it intended by the manufacturers.

The evidence that occasioned this decision has been years in the collecting; the hearings occupied a hundred and forty-two days, and the testimony of 2209 experts, including 47 physicians, was made a part of the record. May the cleanup of this widely publicized patent medicine, if the decision is upheld in the court of appeal, be the forerunner of similar things to come!

Homoeopathic Advocate and Guide to Health.— A new Journal has been received from Keene, N. H., with the above significant title, and which is, we presume, destined to do its part in revolutionizing the whole world in the principles and practice of medicine.

Boston M. & S. J., *April 23, 1851*

OBITUARY

DR. WILLIAM BRADFORD ROBBINS

Dr. William Bradford Robbins, beloved Boston physician for many years, died on January 16, 1951, at his home at 356 Marlborough Street.

Doctor Robbins was born in Boston, the son of Chandler Robbins, D.D., pastor of the Second Church in Boston, and Sarah Ripley Fiske Robbins. He graduated from Harvard College in 1899; his twin brother Tom and his brother Chandler were also members of the same class.

He and his brother Chandler graduated from Harvard Medical School with the class of 1904. Bill, as he came to be called by all his mates, made friends readily; he early became a member of the Boylston Medical Society. He was elected to the third chapter of the Aesculapian Society, and always took a very active interest in its affairs. He was treasurer from 1909 to 1913 and president in 1914, rarely missing a meeting.

He married Marian S. Bennett of Wayland in 1904. They had three daughters and three sons, the youngest of whom, Peter, follows in his father's footsteps and is now at the Peter Bent Brigham Hospital.

Dr. Robbins was appointed a medical intern at the Boston City Hospital, and after completing his service there began the general practice of medicine in Boston. In due season he was appointed to the medical staff of the Massachusetts General Hospital, and at the time of his retirement had been a visiting physician there for several years. His keen sense of humor and his humanitarian approach to medical problems made his ward rounds a stimulating experience for his house staff and nurses. His cheerful "Have a Lifesaver" eased many a tense situation.

Although he liked his hospital service and took it very seriously, his real love was his private practice. This became large and exacting as devoted families of patients grew into second and third generations. His patients were his friends. No service was too menial, no effort too great. He might motor sixty miles to rescue a patient from a hurricane or flood or to listen patiently to real or imaginary complaints with equal sincerity.

No matter how busy he was he had an air of leisureliness about him, so his patients never felt hurried in discussing either their symptoms or a recent fishing trip. His cheerful smile, ready wit and ability to say the right word at the right time left an aura of hopefulness with many "shut-ins" long after he had left them. Sympathetic, understanding, wise in counsel, he exemplified to a high degree the truly devoted family physician. His Christian faith, his humility and his kindness endeared him to all his friends and patients.

He wrote in his fiftieth class report that he had spent his life in trying to keep his friends well and that his "enduring satisfactions" were his wife and children.

Although devoted to his profession, he loved outdoor life. He liked to hunt quail in Tennessee. He enjoyed fishing and golf, and was proficient in both. He was a jolly companion and a good sportsman.

He loved his summer place in the congenial doctors' colony along Argilla Road in Ipswich. Here he enjoyed such leisure as he could snatch from his busy practice, enjoying the view, playing a round of golf and visiting friends — always a welcome guest and a jovial host.

The large number of classmates, friends and patients who filled Trinity Church and sang with his spirit the hymns sung so many Sunday evenings at Ipswich showed the esteem in which he was held. Surely it may be said of him:

> Well done, good and faithful servant;
> Enter thou into the joy of the Lord.

W. G. P.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS

The May schedule for Consultation Clinics for Crippled Children in Massachusetts under the provisions of the Social Security Act follows:

Orthopedic Clinics	Date	Clinic Consultant
Haverhill	May 2	William T. Green
Greenfield	May 3	Charles L. Sturdevant
Lowell	May 4	Albert H. Brewster
Salem	May 7	Paul W. Hugenberger
Gardner	May 8	Carter R. Rowe
Brockton	May 10	George W. Van Gorder
Lynn*	May 14	Daniel M. Killoran John J. Crowley
Springfield	May 15	Garry deN. Hough, Jr.
Worcester	May 18	John W. O'Meara
Hyannis	May 24	Paul L. Norton
Fall River	May 28	David S. Grice
Rheumatic Fever Clinics		**Dates**
North Reading		May 1, 8, 15, 22, 29
Fitchburg		May 2, 9, 16, 23
Plastic Clinics		**Consultant**
	Semi-monthly	
Eastern Massachusetts		Bradford Cannon
	Monthly	
Western Massachusetts		Joseph M. Baker

Physicians referring new patients to clinics should get in touch with the district health officer to make appointments. Patients are seen by appointment only.

*This clinic serves Lynn, Lynnfield, Saugus, Nahant, Revere, Chelsea, Everett and Winthrop. Referrals should be made directly to the Lynn Hospital.

CORRESPONDENCE

PAYMENT UNDER PROTEST

In December, 1950, Dr. Leland S. McKittrick, president of the Massachusetts Medical Society, addressed a letter to all fellows of the Society who had failed to pay their dues for 1950 to the American Medical Association. The following correspondence ensued. It is published here with the permission of its authors.

December 29, 1950

Massachusetts Medical Society
8 Fenway
Boston

Gentlemen: As a result of Dr. McKittrick's excellent letter I am enclosing check for $25 for my American Medical Association assessment. I do so because payment of this assessment has been ruled as necessary for members by the representatives of the profession, and, as a believer in democracy, I must accept the rule of the majority, even though I don't like it.

However, I should like a strong protest to accompany my check and, in the hope that the protests of many of us will reach the House of Delegates, I am sending copies of this letter to each of the delegates from the Massachusetts Medical Society. It is most heartening that such a large proportion of the members of the Massachusetts Medical Society have failed to pay the $25 because it indicates widespread dissatisfaction on the part of the rank and file of the profession with the policies and tactics of the national organization. My own dissatisfaction arises on two main counts:

1. Fundamental disagreement with a basic tenet of American Medical Association policy that only doctors are competent or even entitled to plan the organization of medical care. I would agree that only doctors can see the professional problems from the medical point of view, but to exclude the consumer of medical care from a voice in how it may best be organized seems very strange.

2. Fundamental disagreement with the tactics that the American Medical Association has followed for a number of years of working *against* "socialized medicine" instead of *for an aggressive program* of its own.

Organized medicine should provide the leadership in improving medical care as a social institution. It ought to be leading the fight for wise bills in Congress and working to draft legislation, instead of running the most expensive lobby in Washington to block medical-care legislation. My reasons for this feeling are twofold: (a) The profession has a moral obligation to provide such leadership; (b) strategically it will do more to stop a comprehensive system of government medicine than the present tactics, which, although they may win a battle, will probably lose the war and thus hasten the coming of the socialized medicine to which we are all opposed. In general, we find ourselves repeatedly supporting programs to which we were bitterly opposed several years before, but only when it's too late and we've acquired the reputation of being obstructionists.

The reversal of the American Medical Association's position from favoring to disapproving Federal aid to medical schools, even though followed by provision of $500,000 (a mere drop in the bucket) for medical education, was a terrible blunder. The ostensible reason was that this was the entering wedge of socialized medicine and that the Federal money would only come with strings attached. Is the American Medical Association money to be given with no strings attached? I fear that two years hence the American Medical Association will be in favor of Federal aid to medical education, when it has become a *fait accompli*.

I am personally vulnerable. I have not been a regular attendant at the meetings of my district medical society, and thus have no right to criticize those who have worked in what they consider to be the best interests of the profession and of the public. However, I do hate to see the organization that represents our profession nationally and that has done such a superb job for the American people on so many of the technical aspects of medicine get the reputation, which it has certainly acquired, of being against all social change. It should be the leader in promoting wise programs for spreading the benefits of modern medicine as widely as possible — programs based on the conclusions of careful studies of needs carried out in co-operation with the general public and with the representatives of governmental and private agencies.

Charles A. Janeway, M.D.

Boston

January 16, 1951

Dear Dr. Janeway: I appreciate your frank letter. I wish there were more like yourself who would pay their dues and continue to stay in the medical societies and perhaps play an effective role in formulating the policies of the American Medical Association.

I agreed with Howard Means that the Association "needs new and more enlightened leadership," but I now say we are getting it. The tired old men who ruled for years are being replaced by young, vigorous men who think clearly. Look at the Board of Trustees: Gunnar Gundersen, Blasingame, Larson and Louis Bauer; Austin Smith, the new Editor; the Council on Medical Education and Hospitals, from W. S. Middleton to Donald Anderson; the Council on National Emergency Medical Service includes Perrin Long and Stafford Warren; the Council on Industrial Health includes Paul Magnuson, A. J. Lanza and W. F. Draper; the Council on Pharmacy and Chemistry includes Ceiling, Harry Beecher and Paul Cannon — incidentally, you know that this Council was started years ago against great opposition but is now recognized as a protection for both the consumer and the doctor.

We need you there too.

Your dissatisfaction with the American Medical Association, you say, rests on two points:

"1. Disagreement with the basic tenet of Association policy that only doctors are competent or entitled to plan the organization of medical care." In reply may I point out the *present-day American Medical Association policy*. The Council on Industrial Health had established a new committee with Labor and Management for the improvement of existing health services in industry. I understand that no effective steps could be taken because of the opposition from labor groups.

The standards of approval for laymen-sponsored medical-care plans were elaborated by a subcommittee of the Council on Medical Services including Dean Clark, Jerry Vorhees and Mr. Crownhart of Wisconsin. These standards were approved by the Delegates in June, 1949, and are still being

reviewed. As you know, there is a lay group organized in Washington, D. C., that now has the approval of the district medical society and is being considered for approval by the Council. The American Medical Association is quite aware of this problem and realizes that some want approval only by the national association. However, there still are others who believe that final approval of permitting doctors to work in an organization should be settled at the state level, which is not American Medical Association business but a local matter.

You also know that many medical-care plans sponsored by state medical societies have adequate representation of nonprofessional people. In Massachusetts nonmedical members comprise two thirds of the Blue Shield governing body.

Next, see, beginning on page 661 of the *Journal of the American Medical Association* of October 21, 1951, the eleven-point program of the Committee on Rural Health that was developed out of the first four National Rural Health Conferences that the Association sponsored; this program is working steadily toward solving the problems through community efforts. The 1950 conference brought together more than 750 farm and health leaders, including representatives of health and farm groups, agricultural extension organizations, chairmen of state rural health committees, deans of medical schools and public-health officials.

"2. Fundamental disagreement with the tactics that the American Medical Association has followed for a number of years working *against* instead of for something." I quite agree with you and I have become involved in American Medical Association affairs in the past two years because of this same feeling. I think the tide is turning — energy expended in tactics born in fear and conceived to defeat a politically ambitious group (the Federal Security Administration under Oscar Ewing) in Washington can now be turned toward the cause that every doctor has at heart — "to render better medical care at reasonable cost." The fighting really has been about the economics. De Voto, the ill-paid writer (*Harper's* should be ashamed of refusing him a cost-of-living raise since 1946), touched the heart of the problem on the last page when he said that "desperate social problems have to be solved." Doctors stand ready and are trying to help solve them, but the battle has been one of costs — and many other components. ***Will the cost be greater under the government-insurance and handout scheme or will it be less if local communities encourage insurance through all means and the present system continues and improves?*** — that is squarely the issue. I personally favor the last. So does the American Medical Association. Back in the 1930's the Association did oppose voluntary insurance, but *today their policy* is to push all forms of insurance or prepayment-of-medical-care plans. May I again refer you to the printed report to the Massachusetts Council from the Delegates meeting in San Francisco in June, 1950 — Supplement No. 1 of the *New England Journal of Medicine*, page 23. The report reads: The Council on Medical Services called a general conference on voluntary health insurance in Chicago June 4, 1950, and agreed "that many agencies are required to extend voluntary prepayment medical and hospital benefit insurance." The Permanente Plan is described.

Now just a word about the "most expensive lobby in Washington." The American Medical Association does maintain a registered lobby in Washington. The Washington office, under Joe Lawrence, collects information on all matters favorable and unfavorable to national health and mails out regular letters to interested doctors and runs a column in the *Journal of the American Medical Association*. Do you want to receive it and keep informed? I am enclosing one of his weekly letters. Does that make you a lobbyist? Last year this office cost the American Medical Association (see *Journal of the American Medical Association*, July 15, 1950, page 986) $87,523.21. Against this the Council on Medical Education and Hospitals spent $193,800.49. Now, through this listening post, we were able in 1949–1950 to be *for something*.

1. The American Medical Association supported the National Science Foundation Bill.

2. The House of Delegates pushed the matter of grievance committees to hear complaints of the consumer.

3. The American Medical Association helped and almost entirely planned the "doctor draft law." E. D. Churchill thinks the Committee with Deans and others went too far in obligating medical students to serve later in the Veterans Administration Hospitals where persons with nonservice-connected disabilities are cared for. (See *J. A. M. A.*, November 25, page 1112, or *The Boston Herald* of December 9).

4. Continued support of the Hill–Burton Hospital Construction Act.

5. The American Medical Association has repeatedly endorsed local public-health units and has testified before committees of Congress in support of bills providing for expansion of local public-health units.

6. The American Medical Association stimulated the establishment of the Hoover-directed report on medical education and the establishment of the National Fund — which should break into print any day, now that we have transfused it with $500,000.

7. The American Medical Association reiterated its support of a Federal Department of Health of Cabinet status and successfully opposed the President's Reorganization Plan Number One, which would have established a Department of Welfare, in which health would have had a subordinate part.

8. The Association stimulated the arrangements for twenty-four-hour medical service at local levels, to answer the criticism of the public.

9. The Inter-Association Committee of the American Medical Association, American Dental Association, American Public Health Association, American Hospital Association, American Public Welfare Association and American Nurses Association was established in 1949. At dinner last night Dean Clark conceded that it was a constructive idea and said that two of three things they agreed on had been accomplished.

10. The Trustees have issued a strong statement *against* overcharging by physicians. Is that bad?

And now, finally, you come to the "terrible blunder" — that of American Medical Association support of medical education. That is squarely on me and the Massachusetts Delegation. But let me clarify the American Medical Association. Its policy has not changed one iota with regard to Federal aid to medical education. Federal aid to medical schools is favored, but not in the form of the bill drawn, which gives control of the situation to the Federal government. Last year I said that I would try to influence the American Medical Association to give money to medical education from its National Educational Fund of $2,300,000 raised to January, 1950. Several of the outside-the-fence shouters laughed at me as being naive in thinking anyone could change those "diehards on Dearborn Street." The fact is that this resolution was introduced in October, 1950, in the Council of the Massachusetts Medical Society and approved by the State Committee on Education. Then, during the Cleveland Delegates Meeting, it was discussed with Dr. Louis Bauer, chairman of the Board, who thought it a fine idea and called a special session of the Board on Tuesday, December 5, and reconvened the House the next morning for the special announcement, which told of the token of our sincerity concerning medical education by pledging $500,000. Then the news reporters tried to distort its intentions, and those with anti-American-Medical-Association factor in their blood look for malicious motives. The greatest hope is that it will start the National Fund for Medical Education and get industrial and financial groups to support us in a fitting manner and keep us out of political control. None of us thought the sum large enough. That is where one quarter of your $25 is going.

Yours for a better world to live in. What do you want us to do next?

EARLE M. CHAPMAN, M.D.
Boston

NOTICES

BOSTON CITY HOSPITAL HOUSE OFFICERS' ASSOCIATION

The Boston City Hospital House Officers' Association will present a lecture as part of its evening lecture series in the Dowling Amphitheatre of the hospital on Tuesday, April 24, at 7 p.m. Dr. Sheila Sherlock, Hammersmith Hospital and Postgraduate Medical School, London, England, will speak on the subject "Carbohydrate Metabolism and the Liver." Discussers will be Drs. Helen W. Deane and Franz Ingelfinger.

All interested persons are invited to attend.

BOSTON CITY HOSPITAL ALUMNI ASSOCIATION

The annual meeting of the Boston City Hospital Alumni Association will be held on Saturday, April 28.

Clinical meetings, with lectures, will be held in the morning. The afternoon program will include visits to the wards, laboratories, operating rooms, blood bank, anesthesia department and the new Joseph H. Shortell Fracture Unit. The annual banquet will be held at the Harvard Club at 6:30 p.m.

The presentation of the General Leonard Wood Medal "for distinguished contribution to medicine and humanity" will be made to Dr. Chester S. Keefer by the president of the Alumni Association, Dr. Max Ritvo. Previous recipients of the medal have been Dr. Elliott P. Joslin, Dr. Frank H. Lahey and Dr. William B. Castle.

HAMPDEN DISTRICT MEDICAL SOCIETY

The annual meeting of the Hampden District Medical Society will be held at the Hotel Kimball, Springfield, on May 1 at 6 p.m.

NEW ENGLAND SOCIETY OF ANESTHESIOLOGISTS

The final meeting of the year of the New England Society of Anesthesiologists will be held in the amphitheatre of Building A, Boston University School of Medicine, 80 East Concord Street, Boston, on Tuesday, May 8.

5:30 p.m. Dinner at the Hotel Somerset.

7:30 p.m. Business Meeting. Annual Election of Officers.

8:00 p.m. Scientific Session.

Panel on Anesthesia for Thoracic Surgery. Leo V. Hand, M.D., moderator. Anesthesiologist, New England Deaconess Hospital, Boston.

Richard H. Sweet, M.D., surgeon, Massachusetts General Hospital, Boston.

Norman Wilson, M.D., surgeon, Overholt Thoracic Clinic, Boston.

Meyer Saklad, M.D., anesthesiologist, Rhode Island Hospital, Providence.

Philip D. Woodbridge, M.D., anesthesiologist, Franklin County Hospital, Greenfield, Massachusetts.

AMERICAN COLLEGE OF SURGEONS

The last of a series of seven sectional meetings of the American College of Surgeons will be held in Detroit, Michigan, on May 9-11, with headquarters at the Book-Cadillac Hotel. Attendance will be largely from Michigan, Ohio, Manitoba, Ontario, Quebec and Saskatchewan, although there is no geographic restriction. Previous meetings were held in St. Louis, January 22-24; Hot Springs, Virginia, February 26 and 27; Philadelphia, March 5-7; New Haven, March 16 and 17; Portland, Oregon, March 26 and 27; and Denver, April 5-7.

SCHOOL PHYSICIANS' ASSOCIATION

The annual meeting of the School Physicians' Association of Massachusetts will coincide with that of the Massachusetts Medical Society and will be held on the afternoon of May 23 at the Hotel Statler (Parlor B).

PROGRAM

1:30 p.m. Business Meeting — open to members only.

2:30 p.m. Feet and Knees. Dr. William T. Green.

3:30 p.m. Practitioner of the Year. Dr. Dean S. Luce.

3:45 p.m. Subclinical Epilepsy in Children. Dr. William G. Lennox.

Booth 86 has been retained and Exhibit 61 of the American Medical Association entitled "Examining the School Child" will be on display.

PHYSICIANS' ART EXHIBIT AT A.M.A. MEETING

The American Physicians Art Association will have its annual exhibit during the American Medical Association convention at Atlantic City, New Jersey, June 11 to 15. Any physicians in the United States, Canada and Hawaii desiring to participate should communicate with the association secretary at 760 Market Street, San Francisco 2, California.

Two hundred trophies will be awarded, as well as a special Helser Trophy — a large decorative cup, which will be awarded for art work done in any medium. A popularity trophy will be awarded to the owner of the entry receiving the most popular votes during the A.M.A. convention.

The Annual Art Banquet will be held Tuesday evening, June 12, at the Marlborough-Blenheim Hotel, Atlantic City, New Jersey.

AMERICAN NEUROLOGICAL ASSOCIATION

The seventy-sixth annual meeting of the American Neurological Association will be held at the Hotel Claridge, Atlantic City, New Jersey, from June 18-20, 1951.

SOCIETY MEETINGS AND CONFERENCES

APRIL 21. Massachusetts Association of Medical Technologists. Page 494, issue of March 29.

APRIL 23. New England Cardiovascular Society. Page 575, issue of April 12.

APRIL 24. New England Deaconess Hospital. Page 576, issue of April 12.

APRIL 24. Norfolk District Medical Society. Page 629, issue of October 19.

APRIL 24. Boston City Hospital House Officers' Association. Page 613.

APRIL 25. Naval Reserve Volunteer Medical Unit No. 1-1. Page 535, issue of April 5.

APRIL 27. New England Deaconess Hospital. Page 576, issue of April 12.

APRIL 28. Boston City Hospital Alumni Association. Notice above.

APRIL 28. American Psychosomatic Society. Page 198, issue of February 1.

APRIL 28. Alumni Association, Long Island College of Medicine. Page 458, issue of March 22.

MAY 1. Hampden District Medical Society. Notice above.

MAY 7-11. National League of Nursing Education. Page 384, issue of March 8.

MAY 8. New England Society of Anesthesiologists. Notice above.

MAY 9. Norfolk District Medical Society. Page 629, issue of October 19.

MAY 9-11. American College of Surgeons. Notice above.

MAY 10. Differential Diagnosis and Treatment of Poliomyelitis. Dr. Louis Weinstein. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

MAY 16. New England Obstetrical and Gynecological Society. Hillcrest Country Club, Leicester, Massachusetts.

MAY 16-18. American Association of Genito-Urinary Surgeons. Page 160, issue of January 25.

MAY 21-25. International Society of Orthopaedic and Traumatic Surgery. Page 422, issue of March 15.

MAY 22-24. Massachusetts Medical Society. Annual meeting. Hotel Statler, Boston.

MAY 23. School Physicians' Association. Notice above.

MAY 24-26. American Goiter Association. Page 384, issue of March 8.

JUNE 7-10. American College of Chest Physicians. Page 494, issue of March 29.

JUNE 9 AND 10. American Society for the Study of Sterility. Page 274, issue of February 15.

JUNE 11-15. American Medical Association. Annual Session. Atlantic City, New Jersey.

JUNE 11-15. Physicians' Art Exhibit at A.M.A. Meeting. Notice above.

JUNE 15-17. American Electroencephalographic Society. Page 346, issue of March 1.

JUNE 18-20. American Neurological Association. Notice above.

JUNE 20-23. American Orthopaedic Association. Page 422, issue of March 15.

SEPTEMBER 4-8. American Congress of Physical Medicine. Page 238, issue of February 8.

SEPTEMBER 19. American Medical Writers' Association. Page 1066, issue of December 28.

SEPTEMBER 19-21. Mississippi Valley Medical Society. Page 1066, issue of December 28.

SEPTEMBER 30-OCTOBER 2. New Hampshire Medical Society. Annual Meeting. Equinox House, Manchester, Vermont.

OCTOBER 31. New England Obstetrical and Gynecological Society. Hotel Somerset, Boston.

NOVEMBER 10-21. Pan-Pacific Surgical Association. Page 680, issue of October 26.

JULY 8-11, 1952. Thirteenth British Congress of Obstetrics and Gynaecology. Page xv, issue of December 14.

(Notices concluded on page xiii)